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GUY'S HOSPITAL REPORTS.

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 Taylor, M. Bramley, Guy's Hospital
 Taylor, W. P., Guy's Hospital
 Tebbitt, E. R., 21, Hopton Road, Streatham, S.W.
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SPLENOMEGALIC

CIRRHOSIS OF THE LIVER :

A SEQUEL.

BY FREDERICK TAYLOR, M.D.

IN a recent volume of these Reports¹ I published three cases of cirrhosis of the liver in children. Two of these had died, and the results of the post-mortem examinations were appended. The third was still living, but he has since died, and I propose to add what is necessary to complete the history of his case.

The report hitherto published is as follows :—

CASE 3.—*Enlarged liver, and very much enlarged spleen ; slight jaundice ; clubbing of fingers ; stunted growth ; epistaxis.*—James W—, æt. 12, was admitted under my care on January 27th, 1896.

He comes from the Gordon Boys' Home. He has never seen his father, and he remembers seeing his mother only three times. He has no brothers or sisters.

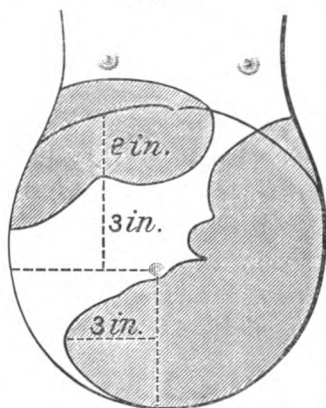
Three and a half years ago he had scarlet fever, and soon after this his illness began. He had pain in the left side, and diarrhœa, but no vomiting. Then his skin became yellow, and jaundice was recognised. After a month the diarrhœa ceased, and he continued well until June last, when pain and diarrhœa returned. He was then in this hospital under Dr. Pitt's care, and went out relieved. Pain recurred a fortnight ago.

¹ Guy's Hospital Reports, vol. lii., 1895, p. 45.

He is very short, and young-looking for his age; height about 4ft.,² weight 4st. 4lbs.; cheerful; not very well nourished. He has well marked, but not intense jaundice. The abdomen is prominent, and the veins are unduly visible. The liver is enlarged, its dulness extending from the upper border of the sixth rib to two and a half inches below the ribs; the edge is half an inch above the umbilicus level in the nipple line, and runs upwards and to the left till it touches the spleen, two inches below the left costal margin. The surface is hard, and to my touch slightly roughened, at any rate not absolutely smooth.

The spleen (Fig. 1) forms a mass extending from the left costal

FIG. 1.



margin to the pubes, so that neither upper nor lower extremity can be felt. Its posterior margin is in the left flank, two inches behind the posterior axillary line. The anterior margin crosses the median line one inch and a half below the umbilicus, and is one inch to the right of the middle line at the pubes. It presents two notches, one just above and to the left of the umbilicus, the other an inch below the umbilicus. There is no ascites. A soft systolic murmur is heard at the apex of the heart, and the pulmonary second sound is accentuated. The finger ends are somewhat thickened, and the hands are very deeply pigmented. The urine contains bile-pigment, and is free from albumen. There are no enlarged glands.

² In September, 1896, his height was 4ft. 1in. It was probably the same in January, as he did not seem to have grown in the interval.

A blood-count on February 7th showed hæmoglobin normal; red corpuscles 80 per cent.; no excess of white corpuscles.

27th.—Hæmoglobin 90 per cent.; red corpuscles 84 per cent. No excess of white corpuscles.

March 7th.—Epistaxis.

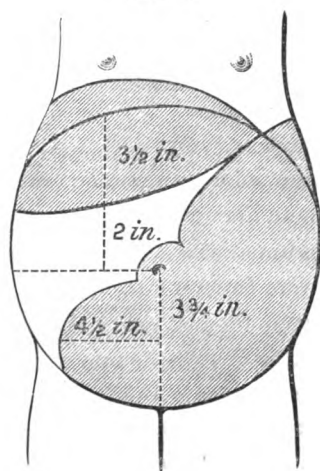
In April the jaundice became decidedly less, and in other respects he was much the same; but the rough and granular condition of the surface, and especially of the edge of the liver, was very pronounced.

The motions, which some months previously had been deficient in the usual fæcal colour, acquired again a normal tint.

He left the hospital on May 13th, 1896, but he was in the hospital from September 18th to October 28th, 1896, and again from May 29th to July 14th, 1897. On the latter occasion he was deeply jaundiced, with spongy and bleeding gums, cutaneous hæmorrhages on the legs, and deep pigmentation of the skin of the abdomen and of the hands. The heart's impulse was below the nipple, in the fifth space, and a systolic murmur was heard there, but was not carried into the axilla. The pulmonary second sound was accentuated.

The condition of the spleen and liver on July 3rd is represented in the accompanying figure (Fig. 2). He was not materially

FIG. 2.



different on his discharge. He was finally admitted under my care on August 9th, 1898, being then fourteen years of age. He had begun to feel worse again about two weeks previously.

On admission.—He was deeply jaundiced. The abdomen was much distended, the umbilicus nearer to the pubes (six and one-eighth inches) than to the ensiform cartilage (seven and a half inches). The veins above the umbilicus were very prominent. The spleen occupied the greater part of the left half of the abdomen, and encroached considerably on the lower part of the right side. The liver was felt three inches below the right costal margin, and the left lobe touched the spleen. There was very little ascites. The lower part of the thorax was bulged outwards by the abdominal contents.

About one inch below the umbilicus could be felt a continuous thrill, no doubt venous in origin, and corresponding to this a continuous hum could be heard with the stethoscope. The heart's apex was in the fifth intercostal space in the nipple line, and a blowing systolic murmur was heard at this point. The red corpuscles numbered 3,000,000 and there was no leucocytosis.

On August 27th it is noted that the abdomen was much distended, the feet were œdematous, and the urine contained a trace of albumen. On September 1st he had some bleeding from the gums.

The quantity of fluid in the abdomen gradually increased, he became apathetic and drowsy, and occasionally had an attack of vomiting or of diarrhœa. On September 13th he was tapped and six pints of serum were withdrawn; after this the liver was felt to be very rough and nodular.

On the 27th it was noted that the urine contained bile-pigment, and that the fæces were free from it.

On September 29th, nine pints of serum were removed by paracentesis, and six and a half pints on October 6th. After this the liver and spleen were well felt, and it was observed that the spleen did not reach over the middle line, as it had done formerly. He gradually sank and died on October 9th.

The following is the account of the post-mortem examination made by Dr. Perry: The boy was wasted, and the skin was deeply jaundiced. On the front of the body the colour of the jaundice was almost black.

The cervical glands were very large and red, and in one of them was some caseous material, and the lymphatic glands throughout the body were enlarged and reddened.

The thymus and thyroid were healthy.

In the upper lobe of the right lung was a small circumscribed hæmorrhage, and below there were a few areas of collapse.

In the left lung were a good many hæmorrhages of considerable size.

The bronchial glands were large and red.

The larynx, trachea, bronchi, mediastinum, pericardium, and heart (which weighed six ounces) were normal.

The veins at the lower end of the œsophagus were not enlarged or varicose. The umbilical vein was, however, persistent and large enough to admit a big probe, and the veins beneath the peritoneum in the hypogastric region were very large and tortuous.

The peritoneal cavity contained a pint or more of bile-stained serum.

The stomach showed slight congestion of the mucous membrane but no submucous hæmorrhage.

The liver weighed forty ounces. It was rough and nodular on the surface, and on section it was hard, tough, and very decidedly cirrhotic.

The gall-bladder contained a little colourless bile, and there were a few adhesions round the gall-bladder.

The pancreas was healthy.

The spleen weighed eighty-seven and a half ounces. There was no change in its obstruction obvious to the naked eye. It did not contain any white nodules, nor was it of "hard-bake" type.

The kidneys weighed fourteen ounces, and were somewhat large, but normal.

Histologically the structure of the kidney was normal.

The spleen was loaded with red blood corpuscles; otherwise it appeared normal.

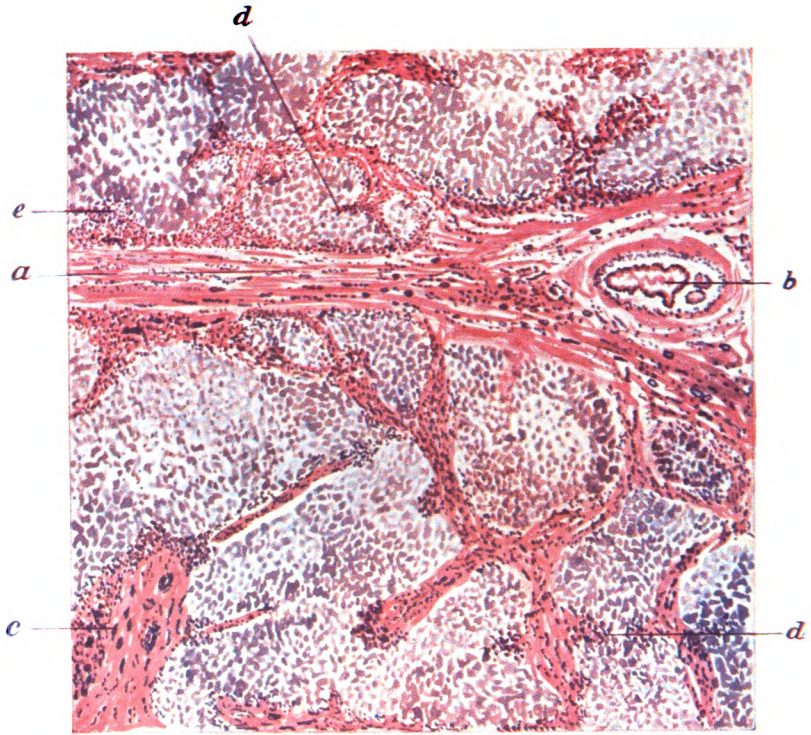
A lymphatic gland contained many cells filled with brown blood-pigment, but was otherwise normal.

I am indebted to Dr. T. G. Stevens for the following report of the microscopic appearances of the liver, and for the coloured drawing which accompanies this paper (see Plate I.) :—

‘The pieces to be examined were embedded in paraffin, and sections were cut and stained with hæmatoxylin and counterstained with fuchsin S. and orange G. The liver substance is seen to be everywhere divided up into well-marked areas by increased formation of fibrous tissue in Glisson’s capsule. For the most part these areas are multilobular—that is to say, many original liver lobules are included in an area. But in places it can be seen that the areas are much more subdivided by the new fibrous tissue, and thus single lobules of the liver become isolated, and even small groups of liver-cells are cut off here and there from the rest of the lobule. The older bands of fibrous tissue (*a* in coloured plate), which for the most part run at right angles to the surface of the liver, are very thick and dense. They contain large numbers of blood-vessels, the walls of which are so thin that they seem to consist only of a single layer of endothelium. In addition to blood-vessels these older bands contain bile-ducts of various sizes in large numbers, the largest ducts (*b*) being obvious spaces lined by long columnar epithelium, the smallest being only a double row of cubical cells with well-stained nuclei. This formation of bile-ducts is a well-marked feature throughout the liver. Everywhere in the new fibrous tissue large numbers of ducts can be seen clearly marked off from the connective tissue. Branching out through the liver substance from the main fibrous tissue, bands are to be seen—bands of various thicknesses corresponding with their age—the older ones having a core of dense fibrous tissue with several layers of small round cells outside (*e*), the younger ones consisting of the round cells (*d*) and only a small proportion of

Splenomegalic Cirrhosis of the Liver.

PLATE I.



Magnified 18 diameters

THOS. G. STEVENS, Del.

- a.* Main fibrous-tissue band, with large numbers of thin walled blood-vessels and many bile-ducts.
- b.* Large main bile-duct.
- c.* New bile canaliculi in new fibrous tissue.
- d.* Connective tissue and round-celled infiltration advancing into the lobules.
- e.* Young connective tissue and round-celled infiltration along the margin of the fibrous band.

embryonic connective tissue. Along the margins of all these fibrous bands can be seen here and there isolated liver-cells cut off from the lobule, sometimes in masses of five or six, sometimes singly. The formation of bile-ducts (c) is most marked in the younger bands of connective tissue. The liver-cells are for the most part unaltered throughout, fatty changes being conspicuous by their absence. The capillaries in some of the lobules are considerably dilated, but this is not a marked feature."

The points of interest in this case have been already discussed in my former paper, pp. 60—64, and I there showed that it belonged to a group of cases which were described by Messrs. Gilbert and Fournier as presenting the following characteristics: Enlarged cirrhotic liver, very much enlarged spleen, jaundice, stunted growth, and clubbing of the fingers. All these features were present in my case. The total duration of the case, as evidenced by jaundice, appears to have been six years, the enlargement of the organs continued to the end, and ascites occurred during the last few weeks. The post-mortem examination demonstrated the correctness of the diagnosis, namely, that the condition of the liver was indeed cirrhosis, as had been believed from an early period of our acquaintance with the case, but no further light has been thrown upon its origin.

Since the publication of this case, some others have been recorded. Two years ago Dr. Hugh R. Smith brought before the Clinical Society³ "seven cases of cirrhosis of the liver occurring in children and young adults," and in one of these (case 5) the conditions were similar to the present, namely, large liver, large spleen, jaundice, stunted growth, and clubbing of the fingers. A case has been recently reported at the Société Médicale des Hôpitaux in Paris, and is alluded to later.

The pathology of the disease.—As was shown in my former paper, and is obvious from all modern writings on cirrhosis, it is a subject by no means so well understood as it was thought to be some years ago, when the irritant action of alcohol upon the

³ Clin. Soc. Trans., vol. xxxi., 1898, p. 258.

hepatic tissues, and the mechanical effects of fibrosis upon the portal circulation summed up the whole of its ætiology and pathology.

While it is certain that many cases of cirrhosis, with large liver and jaundice, are due to alcohol, it must be allowed that a non-alcoholic cirrhosis has to be reckoned with, and the more one looks to the possibility of a general infection (*e.g.*, scarlatina or pneumonia) rather than a local poisoning, as that of alcohol has been so long regarded, the more it seems likely that disturbances of function may occur in parts other than these under the immediate control of the liver and its circulation.

From this point of view the condition of the spleen itself has to be considered. We are familiar with the fact that in the more usual cases of multilobular and atrophic cirrhosis with ascites, the spleen is enlarged, and the enlargement is attributed to the obstructed portal circulation damming back the blood in the splenic vein, a process analogous to that by which the ascites is produced. But this is only a moderate enlargement, whereas in a case like the present the spleen is enormously increased in size (eighty-seven ounces in this case), while ascites is commonly entirely absent until quite the end of the history. It is difficult to believe that any obstruction in the portal system should act in such a partial manner upon its respective tributaries, the splenic and mesenteric veins, as to produce this enormous engorgement in the case of the spleen, and absolutely no effect upon the peritoneum.

The cause of the splenic enlargement cannot be purely mechanical. The histological changes in this case described by Dr. Perry were those of a normal organ, engorged with blood. To produce such a condition in a spleen from sixteen to twenty times its normal bulk, there must have been a considerable degree of actual hypertrophic growth.

The relation of the liver to the spleen in these cases even now excites a good deal of discussion in Paris, of which the brief records may be seen in the current numbers of *La Semaine Médicale*.

At the sitting of the Société Médicale des Hôpitaux on April 6th, MM. Milian and Landrieux reported⁴ the case of a man whose illness began last October with pains in the left hypochondrium, and alternating diarrhoea and constipation. A considerable hypertrophy of the spleen was observed, and some time afterwards slight jaundice and enlargement of the liver. The patient died in February from hæmatemesis, which was found to proceed from an œsophageal varix. The diagnosis was Hanot's cirrhosis, with splenic origin (*à début splénique*). The autopsy showed hypertrophic biliary cirrhosis, a protobiliary cirrhosis, with periangeiocholitis.

The liver weighed 1900 grammes; the spleen 2700 grammes. All the abdominal glands, especially those in the hilum of the liver, were enlarged. M. Chauffard remarked that this case was in accordance with his view that the splenic lesion often preceded and determined that of the liver. He was unable to regard such a case as coming within the category of Hanot's biliary cirrhosis. M. Hayem said that the case corresponded exactly with the morbid condition he has described under the name "Chronic splenomegalic jaundice"—which he believed differed entirely from Hanot's disease.

At the meeting of the same society⁵ on May 2nd, Mr. Gilbert said that M. Milian's case was a new example of splenomegalic hypertrophic cirrhosis, or, as he should like to call it, *hyper-splenomegalic cirrhosis*.

Again, at the meeting of the society⁶ on May 18th, M. Chauffard spoke on the subject. He objects to Mr. Gilbert's term *hyper-splenomegalic*, and believes that the important points in each case are the relations, in time, of the hepatic and splenic lesions respectively. He recognises these three groups:—

(1). Cases in which the spleen and the liver appear to be affected simultaneously, and in almost equal degree—*splenomegalic hypertrophic biliary cirrhosis*.

(2). Cases in which the spleen is affected markedly before the liver, and in higher degree; he considers these to be cases of

⁴ La Semaine Médicale, 1900, p. 124.

⁵ La Semaine Médicale, 1900, p. 154.

⁶ La Semaine Médicale, 1900, p. 176.

hepatitis of splenic origin, and would call them—*metasplenomegalic hypertrophic biliary cirrhosis*.

(3). Cases in which the hepatic lesion precedes and appears to determine the change in the spleen—*presplenomegalic hypertrophic biliary cirrhosis*.

At the meeting of the 25th May,⁷ Mr. Gilbert expounds his views more fully. He adheres to his term *hypersplenomegaly*, and regards the disease as a variety of biliary cirrhosis, distinct from Hanot's type, and characterised by chronic icterus, by *hypersplenomegaly*, and by moderate hypertrophy of the liver. He points out the frequency of the disease in youth and adolescence, its equal occurrence in both sexes, its independence of acute and chronic infections like syphilis, tubercle and paludism, and of more common intoxications, such as the alcoholic, and its possible origin in the gastro-intestinal tract.

He recognises three kinds of onset—(1) *Gastro-intestinal*, with anorexia, vomiting diarrhœa, or abdominal pains; (2) *Hepatobiliary*, with jaundice progressively deepening, painful sensations in the right hypochondrium, and hepatic enlargement; (3) *Splenic*, in which pain in the splenic region and enlargement of the spleen apparently precede the hepatic phenomena. He expressly points out here not that the cirrhosis arises from the splenic lesion, but that the first symptoms concern the spleen and the splenic region, and that if the spleen really causes the cirrhosis, it must be through the medium of the splenic vein, and the cirrhosis would have then a venous and not a biliary type of histological structure. The frequent hepatic commencement and the occasional acute biliary attacks and the histology of the lesion, favour the view of a biliary origin. Biliary cirrhosis may be accompanied by small spleens, large spleens, or spleens of intermediate size. This is a variety with a large spleen, and in the very large spleen and the relatively small liver it is distinguished from the well-known Hanot's type.

M. Chauffard, in reply, says it must be admitted that the lesion of the spleen precedes in time, but not that it causes, the

⁷ La Semaine Médicale, 1900, p. 186.

lesion of the liver, and the origin of the disease is an intestinal infection.

The above discussion shows how obscure is the pathology of this group of cases, and some of the remarks that follow upon other conditions, such as stunted growth and pigmentation, will accentuate this. One may earnestly hope for an early illumination of our darkness, if only to deliver us from the cumbrous and unwieldy nomenclature which has been suggested for temporary use by our foreign confrères.

It will be seen from the description of the histology of the liver that the distribution of the fibrous tissue was not exclusively multilobular or unilobular. Apparently the multilobular arrangement of the fibrosis was much more pronounced than it is usually admitted to be in cases where the liver is so large, the jaundice so marked, the duration so long, and the ascites so tardily developed as in this case. As a rule, no doubt, these cases have either been shown to belong, or have been regarded as belonging, to Hanot's type—the type of hypertrophic biliary cirrhosis. In Dr. Smith's case there was "great increase of connective tissue, which tends to surround the individual lobules of the liver"; and M. Milian's case, recently mentioned, was one of "hypertrophic biliary cirrhosis." If my case shows that hard and fast distinctions between the varieties of cirrhosis cannot safely be drawn, it is no more than has been recognised for many years already.

Arrest of growth.—The stunted growth of the patients in some of the recorded cases, is a very difficult subject to understand, without a good deal more material than is at present to hand. It may be asked, Were the patients really shorter than normal? Were they of normal growth before the disease commenced? How long must a disease of this kind operate to affect the growth? I think I can safely say that my patient was unusually short. He was said to be 12 years of age on his first admission, and eight months later, when he was probably 13, he was only 4ft. 1in. I know boys of medium growth, not exceptionally tall, who have reached 4ft. to 4ft. 1in., at seven and eight years of age; a tall boy of six years of age was 4ft. high. Some records from a school show 18 boys of 12 years of age

with an average height of 4ft. 9in. (max. 5ft. 3in., min. 4ft. 3in., 39 per cent. 4ft. 7in. or 4ft. 8in.), and 36 boys of 13 years of age with an average height of 4ft. 10in. (max. 5ft. 3½in., min. 4ft. 5½in., 41 per cent. 4ft. 10in. or 4ft. 11in.) It is true we have no legal evidence of Wigmore's date of birth; he was homeless and friendless, and he may have been younger than was stated to us, but he had all the facial appearance and development of a boy of the age stated, and there can be no doubt his height was much below the average. Dr. Hugh Smith's patient (case 5) was 23 years of age, and is stated to have been undersized, and this can, of course, be accepted without question in one obviously an adult.

But another question is, How long was the disease in operation, and at what age did it begin? My patient was first seen at twelve years of age, and stated his first illness to have occurred three and a half years previously, that is, about the age of eight and a half.

Dr. Hugh Smith's patient had scarlet fever at the age of eleven, was ailing from that time, and at thirteen years of age was easily tired, had a dark, itching skin, and frequent epistaxis. At seventeen years of age she was known to have jaundice.

The cases of Drs. Gilbert and Fournier may be tabulated as follows: they are not all individually described as stunted, though there is a general statement to that effect:—

			Age at time of observation.		Age at beginning of illness.		Maximum duration of illness.
Case	1	...	13	...	5	...	8 years.
	2	...	19	...	17	...	2 "
	3	...	13	...	11	..	2 "
	4	...	12	...	11½	...	6 months.
	5	...	10	...	9½	...	7 or 8 months.
	6	..	13½	...	12½	...	1 year.
	7	...	13	...	12	...	1 "

Case 1, at 12 years of age weighed 29 kilo. 400 grammes, or 4st. 8½lbs. (My case weighed 4st. 4lbs. at 12 years old). Case 4 is said to have always been "chétif." Case 5 was "small for his age." Of Case 2 it is clearly stated that he was nineteen

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Splenomegalic Cirrhosis of the Liver

PLATE II.



Skiagram showing bulbous condition of the ends of the fingers.

when observed, that two years ago he had pain in the hypochondrium, and that he was of short stature, namely 1.48 metres, or 4ft. 10½in., and weighed 49 kilog., or 7st. 10lbs. One of two things, either his short stature was entirely unconnected with the cirrhosis, or the disease, whatever its nature, or some causative factor in the disease was in operation many years before. In their first case the illness is said to have commenced at five years old, a time when one can easily conceive an effect to be produced on growth. In all the other cases, that is in their remaining five, and in Dr. Smith's case and mine, the age of the commencement of the disease was from eight to twelve years. It will be interesting if in future cases any further facts can be ascertained bearing upon this point.

In any case the stunted growth must depend on a defect in the growth of bone, however arising. Our French authors call attention to other changes in the bones and joints; ungual phalanges voluminous, swollen and looking like drumsticks; lower end of the femur and the ends of the tibia enlarged; knees painful and containing fluid. The thickening of the finger-ends was a prominent feature in my case, and in Dr. Smith's cases the "fingers are clubbed." But so far as my own case is concerned I can state positively that this was not a change in the bone or the joints, but in the tissues. The skiagram which accompanies this paper shows the absolutely normal outline of the phalanges and the swelling which belongs to the soft parts alone (see Plate II.).

Dr. Kingston Fowler⁸ has published a skiagram of clubbed fingers from a case of bronchiectasis, which also shows that the bones are not involved in the swelling.

Dr. Samuel West⁹ succeeds in showing that nothing is known as to the cause of clubbing of the fingers in general, and that it is not a true œdema, although so often associated with diseases in the chest, obstructing the circulation.

Fluckiger¹⁰ is quoted by West as having recorded a case of clubbed finger-ends in cirrhosis of the liver, but the case scarcely

⁸ Diseases of the Lungs. Fowler and Godlee, 1899.

⁹ Clin. Soc. Trans. Vol. xxx., 1897, p. 60.

¹⁰ Wiener Mediz. Wochensch., 1884, p. 1457.

falls into line with those under present consideration. The patient, a female aged 37, was not jaundiced, but the skin of the whole body was of a moderately intense blue-grey colour, the hands and face in the highest degree. Four months before death she had hæmoptysis, subsequently had mental disturbances suggestive of an early dementia paralytica, and died after several days' drowsiness. Nothing had been found in the lungs, or heart, or great vessels. Post-mortem the heart was found to be normal, but there was enormous dilatation of the veins of the œsophagus, lungs, brain and meninges, spinal cord, and of the veins of the body generally, and especially of the abdominal veins. In addition were found syphilitic lesions on the bones, a round scar on the left labium, and a pronounced cirrhosis of the liver. Fluckiger refers the dilatation of the abdominal and œsophageal veins to the cirrhosis, but is quite unable to explain the dilated veins in the rest of the body and the clubbing of the fingers.

Pigmentation of the skin.—This is another feature of considerable interest in these cases. In my own case the change was especially marked in the fingers, and was quite distinct from the jaundice, with which, at any rate in this part, there was no likelihood of confounding it. The fingers were of a rich brown colour.

The condition has been noted in other similar cases. In the fourth of Messrs. Gilbert and Fournier's cases it is stated that the skin "is of a dirty brown colour, and this tint is uniformly spread over the whole body," and four months later it is noted that the skin is "plus brune, plus bistrée."

In Dr. Hugh Smith's fifth case¹¹ the pigmentation of the skin was one of the first signs noticed. Since the scarlet fever she had been always ailing, and "two years after the attack, when she was thirteen, she was noticed to be weak, very easily tired, and was much troubled with severe itching of the skin, which was also noticed to be dark in colour." This was a different thing from the jaundice, which was first recorded at the age of 17, and was intense at the age of 23. "Skin uniformly tinted a brown-yellow colour, and conjunctivæ are deeply jaundiced."

¹¹ Loc. Cit., p. 263.

The relation of bronzing of the skin to cirrhosis of the liver is discussed in a paper read by Dr. Osler before the British Medical Association¹² at Portsmouth in 1899. From this paper some of the facts in the history of this relation may be gathered—the recognition by Hanot of pigmentation in cirrhosis, the occasional occurrence of diabetes in such cases, the recognition of a diabète bronzé, the occurrence of pigmentation in anæmia (Quinke), and the occurrence of cases of general pigmentation, to which von Recklinghausen gave the name of *Hæmochromatosis*. In these cases not only is the skin deeply pigmented, but large quantities of pigment are found in the liver, pancreas and lymphatic glands. As much of this pigment contains iron (Hæmosiderin), it is believed to be derived from the blood, but some at least is iron-free, and is called Hæmofuscin.

The subject is very fully discussed by Anschutz in an article on Diabetes with bronzing of the skin,¹³ and his conclusions are to the effect that the pigment is formed as a result of a destruction of the blood (Hæmolysis), for which no cause can be assigned, and that the deposition of the pigment in the liver and pancreas causes cirrhosis and diabetes respectively. In none of his twenty-four collected cases was there any jaundice.

Dr. Osler records two original cases of hæmochromatosis with an enlargement of the liver, presumably cirrhosis. In both the liver and spleen were only moderately enlarged, and there was entire absence of jaundice as well as of glycosuria. The patients at the time of record were still living.

I need not go further into this subject. It is clear that my case cannot be classed with those of general hæmochromatosis. That the pigment was derived from destruction of the blood is extremely likely, but that it was causative of the cirrhosis is negatived by the fact that the pigment is not present in the liver. Nor was it observed in the pancreas, but some was found in a lymphatic gland, a not uncommon event. Whether it preceded the jaundice in my case cannot be ascertained, but it is interesting to note that the change of colour of the skin was observed in Dr. Smith's Case 5 and Dr. Gilbert's Case 4 long before the occurrence of jaundice.

¹² Brit. Med. Journ., Dec. 9th, 1899.

¹³ Deutsches Archiv. für Klin. Med., 1899, vol. 62, p. 411.

DISEASES OF THE PANCREAS.

BY W. HALE WHITE.

So little space is devoted to Diseases of the Pancreas in most books upon medicine, that it seemed to me worth while to analyse the experience to be gained from the post-mortem room of Guy's Hospital.

I searched the post-mortem reports for the years 1884 to 1897, both inclusive, that is, for fourteen consecutive years. During that time the number of post-mortem examinations has been 6,708, and the pancreas has appeared to the morbid anatomist diseased or injured in 142 instances, that is, in 2 per cent. of all cases dying in a large general hospital. The following table shows the various morbid conditions found:—

Table showing the number of occasions in which the pancreas was observed to be diseased or injured, in the post-mortem room of Guy's Hospital during the fourteen years 1884–1897:—

Primary malignant disease of pancreas	31
Cirrhotic congested or hard pancreas	26
Small atrophic pancreas	19
Growth adherent to pancreas	13
Secondary growths in pancreas	11
Fatty pancreas	9
Dilated pancreatic duct
(In some cases in which the pancreas was otherwise diseased the duct was dilated, see p. 38, but the cases have not been counted twice over.)				
Tubercle of pancreas	4
Mechanical damage to the pancreas	4
Ulcer of stomach adherent to pancreas	4
Calculi in pancreas	3
Suppuration of pancreas	3
Cysts of pancreas (including one case of hydatid)	3
Hæmorrhage into pancreas	3
Lymphadenoma of pancreas	1
Cavity in head of pancreas	1
Ulcer of duodenum adherent to pancreas	1
Kidney adherent to pancreas	1
Action of acid on pancreas	1

In addition there was one instance of an accessory pancreas. The various diseases and injuries will now be described, and the lessons to be learnt from the post-mortem room of Guy's Hospital will be contrasted with the statements of Fitz in the article, "Diseases of the Pancreas," in vol. 4 of Allbutt's *System of Medicine*, with those by Leo in the article, "Diseases of the Pancreas," in vol. 8 of the *Twentieth Century Practice of Medicine*, with those by Oser in the article, "Die Ekrankungen des Pancreas," in vol. 18 of Nothnagel's *Specielle Pathologie und Therapie*, and with those by Lancereaux in his "Traité des Maladies du Foie et du Pancreas," for these writings may be fairly taken to represent modern teaching on the subject. Each of the headings in the table will be mentioned separately. The individual cases are contained in the appendix (see p. 49).

PRIMARY MALIGNANT DISEASE OF THE PANCREAS.

Histological examinations have not been made sufficiently often for us to be able to give the precise relative proportion between sarcoma and carcinoma, but one case at least (1886, 329) was a small round-celled sarcoma which, growing from the head of the pancreas, involved the duodenum. But as in many instances a histological examination of the growth was made, and as in many others the description of the naked-eye characters of it suggests strongly that it was carcinomatous, we are able to conclude that primary carcinoma of the pancreas is very much more common than primary sarcoma, which is, indeed, quite rare. It is interesting to notice that the solitary patient with undoubted primary sarcoma was a male only twenty-eight years old, that is eight years younger than 1895, 204 the youngest case of undoubted carcinoma, for we are thereby reminded that Drs. Perry and Shaw¹ have shown that in the stomach primary sarcoma occurs at a younger age than primary carcinoma. They found four cases of sarcoma of the stomach among fifty cases of malignant disease of that organ; the ages of the four patients were fifteen, eighteen, thirty-eight and sixty-seven, while the youngest of the forty-six sufferers from carcinoma was thirty-two. In all four of their patients the growth was a round-celled sarcoma, as it was in the patient

¹ Guy's Hospital Reports, vol. 48.

1886, 329, in whom primary sarcoma occurred in the pancreas. Reference to the appendix will show that this patient died very rapidly. She was dead in between seven and eight weeks after the pain was first felt, and on admission, three weeks before death, the tumour was already very large, measuring three by three and a half inches; at death there were secondary deposits in the mesentery, omentum, kidneys and lungs. All these facts suggest that primary sarcoma of the pancreas is a particularly rapid disease met with mostly in young adults. Possibly, as in case 1892, 373, the patient was only thirty-three, and the growth in a short time became as large as a foetal head, this, too, may have been an instance of sarcoma.

Neither Fitz nor Leo give any information on the subject of sarcoma of the pancreas. Oser refers to several cases. He considers sarcoma of the pancreas to be very rare, and believes that secondary sarcomatous growths, especially melanotic sarcoma, are more frequently seen in the pancreas than the primary form which is excessively uncommon. He, however, gives references to several cases. In one an attempt was made to remove the growth, in another recorded by Litten the patient was only four years old. Some of Oser's cases may well be lymphadenomatous rather than sarcomatous. He quotes Segré, who gives the proportion of sarcoma to carcinoma of the pancreas as two to one hundred and twenty-seven.

Lancereaux does not describe sarcoma of the pancreas as such, but under the heading of "*fibromes pancréatiques*" gives an account of Litten's case.

In connection with what has been said about the age of the solitary case of undoubted sarcoma in my series, it should be mentioned that Oser mentions cases in elderly people, but the cases which have been subjected to microscopical examination in recent years are far too few to conclude definitely whether, as my single case seems to suggest, it is a point of distinction between sarcoma and carcinoma of the pancreas that sarcoma usually occurs at a younger age.

As primary sarcoma is so very rare, and the symptoms produced by it so closely resemble those of carcinoma, we may now

pass on to consider primary malignant growths of the pancreas as a whole

The table shows thirty-one cases of primary malignant disease of the pancreas; these occurred among 6,708 post-mortem examinations, that is to say, it is the cause of death in about one in every two hundred of all the patients who die at Guy's Hospital. The table also shows eleven cases of secondary growth in the pancreas, and thirteen in which a growth of some other organ became adherent to the pancreas, so that out of fifty-five cases in which this organ was affected with malignant disease the growth was primary in it in thirty-one. It is interesting to notice that our experience at Guy's is very different from Leo's statements. He says, "Carcinoma is the most frequent disease of the pancreas and may be of primary or secondary origin, the latter being somewhat the more usual. In this case there is generally a direct extension of the carcinomatous process from a neighbouring organ . . .; more rarely the disease arises by metastasis from carcinoma of some distant organ." Fitz makes no reference to this point. Lancereaux merely states that the pancreas is affected secondarily either by direct extension, and then usually from the stomach or duodenum, or by metastasis from some distant organ.

Oser gives the following quotations. Segré, whose cases were Italian, makes the number of cases of malignant disease of the pancreas about 1·12 per cent. of all deaths. Biach, among 23,611 post-mortem examinations, met with twenty-nine cases of carcinoma of the pancreas, that is, in about 0·12 per cent. Eppinger's figures, including 1,314 post-mortems, give the proportion as 1·4 per cent. of pancreatic carcinoma, but only 0·14 per cent. of primary pancreatic carcinoma. Soya found primary carcinoma present in only three cases, or 0·07 per cent. of 3,950 post-mortems. Urany, Foster and Willigk all had too small number of post-mortems for the figures to be of value. At Guy's we have seen that the pancreas was affected with malignant disease in fifty-five out of 6,708 post-mortems, or in 0·8 per cent., and with primary malignant disease in thirty-one cases, or in 0·47 per cent. Leaving for a moment the question as to whether the growth was primary in the organ, we have Segré giving for Italian cases 1·12

per cent., myself giving English cases 0·8 per cent., and Biach giving Viennese cases 0·12 per cent. Why carcinoma of the pancreas should apparently be so much rarer in Vienna than Italy or England is not clear, for even if we assume that among the Viennese cases only those are included which are primary, they are rarer than at Guy's in the proportion of 0·12 to 0·47.

Oser states that secondary carcinoma of the pancreas is more common than the primary form. Thus we see that our experience at Guy's does not correspond with the statements of Leo and Oser on this point.

Olivier (quoted by Oser) believes that histological examination would show frequently that cases of apparent primary malignant disease of the pancreas are really secondary to primary malignant disease of the duodenum. Judging by the naked-eye appearance of cases I have seen I should say that this is very unlikely.

We now pass on to the description of the most important of disease of the pancreas, namely, primary malignant disease (nearly always carcinomatous) of it.

An analysis of the thirty-one cases taken from Guy's Hospital shows that it is commoner in males than females in the proportion of three to one.

With regard to the ages at death, one patient (the case of sarcoma) was under thirty, five were between thirty and forty, ten were between forty and fifty, ten were between fifty and sixty, four were between sixty and seventy, and one was over seventy. As there are fewer people alive of ages between fifty and sixty than between forty and fifty, it is clear that primary malignant disease of the pancreas is a malady which falls most heavily on the decade from fifty to sixty.

The usual duration of the disease is impossible to determine exactly. It is difficult to decide when it began, and the time it lasts must depend upon the variety of carcinoma and the abundance of secondary deposits. Some patients linger on and die from sheer exhaustion, whilst others die early from hæmorrhage or because the growth causes intestinal obstruction, or because for some reason or another an operation is performed; but speaking roughly it appears that the patient is usually dead

within six months of the first onset of the symptoms and that life is rarely prolonged to nine months from the first onset; on the other hand, some patients are dead within three months or even less. On the whole it is a rapid disease.

Abdominal pain is an almost constant symptom and sometimes it is very severe and paroxysmal. It is often associated with tenderness. These facts are interesting in connection with a specimen of cancer of the pancreas which I showed to the Pathological Society² in which the cancer had infiltrated the semilunar ganglia. Probably these and the adjacent nerves are often affected for they lie so near to the pancreas.

Unless some accident carries the patient off quite early in the disease there is nearly always considerable wasting.

Vomiting, too, is a very common symptom; usually it is very difficult to treat; incessant vomiting may so exhaust the patient as to kill him. Occasionally there is a little blood in the vomit, this may be due to rupture of some small vessel from straining, or it may be due to ulceration of the growth directly into the stomach.

Jaundice is present in at least half, and probably more than half the cases. This is not surprising, for the growth is nearly always in the head of the gland, so that the common bile-duct becomes compressed; indeed, so often does the growth surround the common duct that the wonder is that jaundice is not more frequent. Even if the common duct escapes pressure at the head of the pancreas, the patient may be jaundiced because of secondary deposits in the glands in the portal fissure. Cholæmia is sometimes the cause of death.

In a certain number of the cases the pancreatic growth forms a tumour that can be felt during life. This is not remarkable when we remember that in very thin persons the head of the pancreas may occasionally be felt even when there is no new growth in it. In one of the Guy's series of cases the growth in the head of the pancreas formed a tumour large enough to bulge the lower ribs. It is quite impossible to state precisely the number of cases in which the growth can be felt during life,

² Path. Trans., Vol. xxxix., p. 147.

for secondary deposits in the liver and other organs may be mistaken for the primary pancreatic deposit. No doubt the growth in the head of the pancreas would be more often felt were it not that the examination of it is often interfered with by the enlargement of the liver or by ascites.

About a third of the patients have ascites; this is often undoubtedly due to secondary deposits in the peritoneum—indeed, cancer of the pancreas has been mistaken for malignant peritonitis—but in some cases pressure of the primary growth, or of secondary deposits, on the portal vein, may be a contributing factor, but this is not always so, for in a patient under my care there was much ascites, the peritoneum was studded with secondary growths, but the head of the pancreas was free, and there was no sort of pressure on the portal vein. The ascitic fluid may, of course, be bile-stained, and the patient may have to be tapped one or more times before death. In one patient in our series the ascitic fluid was chylous. The autopsy did not show precisely how this had come about. In this case the left pleural cavity also contained some chylous fluid.

In about one-fourth of the cases the account of the post-mortem states that the gall-bladder was distended, but owing to the frequent presence of an enlarged liver, or of masses of growth or of ascites, the enlargement, even when present, cannot often be made out during life. Therefore, as it is only found in the post-mortem room in a quarter of the cases, this very valuable sign, indicating pressure on the common duct, is not often present to help us in our diagnosis. As might be expected, the liver is sometimes enlarged, often because of secondary deposits in it, at other times because the common duct is compressed.

The patient is commonly constipated, and if there is obstructive jaundice the motions may be pale. They may also contain blood. This is not surprising for the growth may invade the duodenum and may bleed into it, and in one case thus caused death.

Bright,³ in 1832, wrote thus, "The symptom to which I refer is a peculiar condition of the alvine evacuation, a portion more or

³ *Medico-Chirurgical Transactions*, vol. xviii., 1833, p. 1.—Cases and observations connected with disease of the pancreas and duodenum.

less considerable assuming the character of an oily substance resembling fat, which either passes separately from the bowels, or soon divides itself from the general mass and lies upon the surface, sometimes forming a thick crust, particularly about the edges of the vessel, if the fæces are of a semi-fluid consistence, sometimes floating like globules of tallow which have been melted and become cold, and sometimes assuming the form of a thin fatty pellicle over the whole, or over the fluid parts, in which the more solid figured fæces are deposited. This oily matter has generally a slight yellow tinge, and a most disgustingly fœtid odour." He records three cases of malignant disease of the pancreas in which this symptom was present, and expressed the belief that the malignant disease of the pancreas was the cause of the symptom, but he records other cases which he says appear to show that it is necessary for this symptom that the growth should have ulcerated into the duodenum, but he does not by any means admit that his cases prove this point, and suggests that the matter should be further observed.

Of the thirty-one cases at the end of this paper, five were under my care but in one the symptom was not looked for, another died shortly after admission, another was very rapid and was considered to be primary malignant disease of the liver, another died quickly from hæmatemesis owing to ulceration of the growth into the stomach, but in the fifth case, as pancreatic disease was suspected, the motions were carefully searched and no fat was found. At the autopsy the growth was found to be in the tail of the pancreas; this is of interest, for Bright records a case in which the growth was in the centre of the gland and the motions were not fatty, and he expressly states that he suggests that the condition of motion he describes is a symptom of malignant disease of the head of the gland. Some authors attach much importance to presence of undigested muscle fibres in the fæces as showing that the pancreatic secretion does not reach the intestine.

Sometimes cancer of the pancreas produces in other organs symptoms so marked that the primary disease is overlooked. This is not uncommonly the case when there are many secondary

nodules in the liver. One of my series of cases presented itself as a case of malignant disease of the colon, for the pancreatic growth had invaded that structure; in another the pylorus was obstructed because the pancreatic growth had extended into it. Another patient was thought to be suffering from some pulmonary affection, which really was the case, for the lung was very full of secondary deposits, and it is interesting to notice that the lung may contain secondary deposits even when there are none in the liver.

Occasionally, as in all cases of cancer, the patient is profoundly anæmic, quite apart from hæmorrhage. In a few instances the muscles on the right side of the upper part of the abdomen are rigid, while those on the left are not; this is a sign of great value as indicating deep disease. The ankles may be swollen, either because of the anæmia or because of the ascites, or from the size of the liver.

Two cases were operated upon in the belief that the symptoms were due to gall-stones, and this mistake in diagnosis is one against which it is well to be on one's guard. In one of the cases there actually were two stones in the common duct, and these were removed, but the patient was also suffering from cancer of the pancreas.

Two patients died from hæmorrhage. In one the growth had ulcerated into the duodenum, in the other into the stomach; such cases lead to hæmatemesis and melæna.

The striking feature of the post-mortem examination is that the growth, which is usually scirrhus, is in the head of the gland in about 85 per cent. of the cases; in the remaining 15 per cent. it is either in the body or tail. The most common organs to be affected secondarily are in order of frequency the neighbouring lymph glands (especially the portal), the liver, the peritoneum and the lungs. Less common seats for secondary deposits are the kidneys, the abdominal walls, the mediastinal glands, the pleura, the pericardium, the heart, the suprarenal bodies, and the kidney.

The growth not infrequently extends into the duodenum and ulcerates it. Less commonly it extends into the stomach, the colon, or the semilunar ganglia.

The bile passages are often dilated, but the pancreatic duct is only occasionally dilated, namely, in four cases out of thirty-one. This is strange, for often the appearance of the parts is such as to suggest very strongly that it must have been compressed by growth. In two of the four cases in which the duct was dilated it was also cystic. In not one of our cases was a pancreatic calculus present, so we may conclude that cancer of the pancreas is nearly always independent of such irritation. In one case the disease was associated with fat necrosis of the omentum.

Contrasting the experience at Guy's with the description given by authors, it appears that we have not met with jaundice or with a dilated gall-bladder so often as Fitz would lead us to expect. He mentions hydronephrosis from pressure of the tumour on the ureter and diabetes as rare results; they undoubtedly may occur, but did not happen to be present in any of our cases. Leo's account of cancer of the pancreas does not contain any statements at variance with the experience at Guy's.

Lancereaux also, I think, lays too much stress on jaundice as a symptom, and like Leo and Fitz hardly lays enough upon the importance of vomiting. He divides the disease into three stages, and considers that the third stage is marked by jaundice and symptoms indicative of compression of the solar plexus. He says that when the growth is in the body of the pancreas it is particularly liable to affect the solar plexus, and that then there may be a bronzing of the skin. He gives a case in point. The patient was wasted and the skin was of a bronzed tint. No details are given as to what parts of the skin were most affected, nor does the author say whether the mucous membrane of the mouth was pigmented. Considering that when the man was first seen he was in the last stage of cancer, and that very often the mistake has been made of confounding the tint produced by cancer of almost any organ with that of Addison's disease, it seems very doubtful whether in this case the bronzing was other than that due to the cancer, independent of its seat, especially as another case of cancer of the pancreas, implicating the solar plexus, is given by Lancereaux, because it reminded him of Addison's disease, although he admits the patient was not

pigmented in a way at all characteristic of Addison's disease. None of the cases I have recorded at the end of this paper in any way support the view that special bronzing of the skin can be produced by malignant disease of the pancreas, and I have in a former paper⁴ brought forward what appears to me to be very strong evidence that disease of the semilunar ganglia and its nerves is not a cause of Addison's disease.

Lancereaux refers to one case in which pressure by a cancer of the pancreas on the superior mesenteric artery obliterated it and so caused intestinal hæmorrhage, and he also, like some other authors, mentions that the presence in the fæces of undigested muscle fibres from the food is a symptom of the disease.

Oser gives a very full and excellent account. He considers scirrhus the commonest form of pancreatic cancer, and certainly that is what we have found at Guy's. He finds that in about 2 per cent. of the cases the growth is melanotic. He mentions that occasionally pressure of the growth on the duct may lead to the formation of cysts in the pancreas; as already mentioned, two cases out of the thirty-one at Guy's showed cystic dilatation of the duct. He devotes much attention to the question of fatty stools, and says that Kuntzmann first described them in association with pancreatic disease in 1820. I do not think there is any evidence that Bright was aware of this when he read his paper in 1832. Unfortunately by a misprint Oser gives the date of the publication of Bright's paper in 1883 instead of 1833. It is quite impossible to give any correct estimate of the frequency of fatty stools in pancreatic disease for so many observers fail to look carefully for them, but Oser does not attach very great importance to them as a symptom of pancreatic disease, for, he considers, they may occur because bile cannot reach the intestine, or as a result of disease of the intestinal mucous membrane, and Sir William Gull⁵ pointed out that fatty stools may occur with disease of the mesenteric glands.

Oser reminds us that the stools are often bulky in pancreatic disease as a result of imperfect intestinal digestion, and that consequently there is frequent constipation.

⁴ Guy's Hospital Reports, vol. xlv., p. 44.

⁵ Guy's Hospital Reports, vol. xvi., p. 369.

He suggests that the pain of pancreatic carcinoma may be like that of locomotor ataxy, for it may be severe and paroxysmal, and he thinks that this is because of the implication of the nerves of the abdominal sympathetic.

His estimate of the frequency of jaundice is about the same as mine, for he thinks it is present in two-thirds of the cases. He thinks that the liver may at first be enlarged from obstruction to biliary flow, and about that there is no doubt, but he thinks that it may subsequently atrophy from biliary cirrhosis. This is possible, but probably very rare, for a cirrhosis due to biliary obstruction and sufficient to cause shrinking of the liver must, I should say, be excessively uncommon. Towards the end of the case the liver may, of course, be enlarged from the deposition of secondary growths, or shrunken as a part of the general wasting.

Oser lays considerable stress upon dilatation of the gall-bladder as a symptom of pancreatic carcinoma, but I have already said I think most authors exaggerate the importance of this sign, for the gall-bladder is only dilated in a quarter of the cases, and as in a large proportion of these it would be impossible (owing to enlarged liver, pancreatic growth, or ascites) to make out the enlargement during life, the sign is of less clinical value than might be expected. Oser, however, well points out that obstruction of the biliary duct in gall-stones is frequently associated with a contracted gall-bladder, whilst if the pancreatic growth presses severely on the duct so as to affect the gall-bladder it is dilated.

He mentions that the ascitic fluid may be chylous, and this is interesting, for it was so in one of our cases. He states that occasionally the pancreatic tumour may move with respiration, and that it may have pulsation transmitted to it from the aorta.

THE CIRRHOTIC, CONGESTED OR HARD PANCREAS.

In twenty-six instances in our series the pancreas has been so described by the morbid anatomist that it may fall under this heading. In seventeen out of the twenty-six cases the patients have suffered from serious organic disease of the heart, and in all but one of these seventeen, namely, 1887, 60, a case of fibroid disease of heart, there was marked evidence of severe backward

pressure from the heart, as shown by nutmeg liver, œdema of feet, tough viscera, &c. The induration of the pancreas was usually very striking: thus we find it described as very hard, or tough; in one case it felt like cartilage. Sometimes the congestion was evident, and in a few cases the organ is described as enlarged. Some cases were examined microscopically and they showed much increase of fibrous tissue. We may, therefore, undoubtedly conclude that long-standing difficulty of venous flow due to disease of the heart will cause induration and fibrosis of the pancreas, as of other organs.

This conclusion makes us suspect that diseases of the lungs and liver may, in the same way, lead to the same result and accordingly we find one case of chronic bronchitis and emphysema (namely, 1889, 251), two of phthisis (namely, 1895, 464, and 1897, 419), two cases of cirrhosis of the liver (namely, 1887, 343, and 1890, 49), and one of thrombosis of the portal vein, in which the pancreas was congested or indurated. Occasionally two causes were present in the same patient, such as disease of the heart and cirrhosis of the liver. In two cases granular kidneys co-existed with cardiac disease, and possibly the induration of the pancreas may in some way be associated with the renal condition, but there is no evidence for such a view. Thus, in twenty-three out of the twenty-six cases the congested or indurated pancreas was associated with and was probably caused by long-standing venous congestion. Of the remaining three cases, one was that of a woman in whom a large ovarian tumour had co-existed with a pregnancy which went to full time; in this case we may fairly assume that the induration was due to direct pressure on the gland. One was a case of diabetes; the pancreas is described as large, but as so many of the patients to whom I shall allude presently as having small and atrophic pancreas also had diabetes, it is likely that this patient, had he lived longer, would have shown a small atrophic pancreas, and that therefore he should be classed in that group. The third case was that of a man, aged 52, who had pleural effusion. This might have caused some backward venous pressure, which perhaps was increased by the severe syphilitic change in the liver, so that perhaps after all

this was a case of hard pancreas due to venous congestion, but I have not put it in that group because it may be open to question whether the tough and hard pancreas, which showed much increase of fibrous tissue and small cells, did not owe its origin to syphilis.

That which I have called the cirrhotic, congested, or hard pancreas, has been described by Leo under the heading of Chronic Interstitial Inflammation (Sclerosis) of the Pancreas. He says, "In the majority of the cases the cause of the interstitial growth lies in an affection of some neighbouring organ which has extended so as to involve the pancreas In other cases the cause lies in disturbances of the circulation and their consequences following disease of the heart, lungs, great vessels, or portal system."

Our experience at Guy's points to the disturbances of circulation being much commoner than the other causes. Leo also mentions "hæmorrhages which not uncommonly result from obstruction in diseases of the circulatory apparatus, the lungs, or the portal system." Certainly such hæmorrhages are very rare in the post-mortem room of Guy's Hospital (see p. 47). Speaking of the causes of sclerosis of the pancreas, he mentions "alcoholism, and especially syphilis"; but apart from affection of the pancreas, secondary to cirrhosis of the liver, we have from the cases on which this paper is founded no evidence that alcohol is a cause of pancreatic cirrhosis, and the evidence in favour of syphilis is inconclusive.

Fitz mentions the form of pancreatic disease we are describing, but gives no account of it. Lancereaux, under the head of hyperæmia of the pancreas, states that disease of heart, lungs, liver, or portal vein, may lead to hyperæmia of the pancreas. He describes a first-stage, in which the organ is gorged with blood and serum, but this is, I think, hardly ever seen; his later-stage corresponds with that which I have described. Under the heading of proliferating or sclerosing pancreatitis he describes alcoholic, malarial, syphilitic, and tubercular pancreatitis. With regard to the alcoholic form, he says it is not rare to observe in a drinker, and particularly if he has cirrhosis of the liver, induration and increase of volume of the pancreas, but he distinguishes

a proper alcoholic chronic pancreatitis, in which the change consists chiefly in fatty swelling of the epithelium, from the induration of the pancreas which may result from venous congestion in cases of cirrhosis of the liver. Under the heading of syphilis he describes a chronic indurative pancreatitis seen in infants affected with congenital syphilis and gummata, but which rarely results from acquired syphilis.

Oser hardly refers to the effect of long-continued backward pressure on the pancreas, but he does mention that occasionally unimportant pancreatic hæmorrhages are due to it. He has a section on chronic indurative pancreatitis, which he divides into inflammation of the whole gland and circumscribed inflammation. The first, he says, may be due to syphilis, alcohol and obstruction of the duct. The syphilitic form may be due to endarteritis, or to a chronic pancreatitis apart from this, when it is nearly always due to congenital syphilis. In the account he gives of the alcoholic form it is by no means clear that the changes in the pancreas may not be secondary to cirrhosis of the liver and consequent pancreatic venous congestion, indeed, in one case he quotes there was thrombosis of the portal vein.

I think there can be little doubt that the authors from whom I have quoted have not clearly in their minds what the cases from Guy's teach, namely, that a hard cirrhotic pancreas is nearly as common as primary malignant disease of the gland, and that it is nearly always due to long-continued backward pressure from disease of the heart, lungs or liver. Further, it appears that if acquired syphilis and alcohol, apart from cirrhosis, can produce chronic pancreatitis, it must be very rare and further investigation upon this subject is required. At Guy's we have no evidence of malarial pancreatitis. In none of the Guy's cases was there any evidence that this condition of gland was due to obstruction of the pancreatic duct.*

SMALL ATROPHIC PANCREAS.

Among our cases of pancreatic disease, nineteen are placed here, and one atrophic pancreas is described with cases of calculi in the

* This paper had gone to press when Mr. Mayo Robson's lecture on Pancreatitis appeared (*Lancet*, vol. ii., 1900).

pancreas (see p. 42). In 1894, 358, the pancreas, which was very firm and fibrous, is not stated to be small, but it is included here as the patient had diabetes. In some of these nineteen cases the pancreas is described as soft and flabby, in others as fibrous and hard; sometimes it weighed only one ounce. Sixteen of the cases had diabetes, and the patient in whom the atrophic pancreas was associated with pancreatic calculi had glycosuria, but died from disease of his heart and lungs. By taking the number of fatal cases of diabetes during the same period, I find that a quarter of the patients dying from diabetes show this variety of pancreatic disease so markedly that it is visible easily the naked eye.

Of the three cases of small pancreas that are not described as having diabetes, in one a large mass of growth pressed on the pancreas; in another, the patient was much wasted and there was no omental fat; and the other was a woman (1892, 44) who died soon after admission, delirious from a severe burn. There is no mention of the state of her urine; possibly had it been tested, sugar would have been found.

Williamson and others have pointed out the great importance of this condition of pancreas and its relationship to diabetes, and our experience at Guy's shows well how frequently the association occurs. Considering all we know of the physiology of the pancreas, there can be no doubt that the condition of the pancreas here described is the cause of the diabetes. It will be remembered that on page 29 we have already referred to the case of diabetes, in which the pancreas was very indurated, but was large, and later a case will be described in which a patient with glycosuria had a cyst which destroyed the whole of the pancreas.

Fitz gives a description of "chronic pancreatitis." He first of all puts aside that due to venous obstruction, and then proceeds to describe a "genuine chronic fibrous pancreatitis." He does not think alcohol and syphilis are causes of it, and considers that "the most probable cause is a chronic catarrh of the pancreatic duct continued from the duodenum into the pancreas." He then

describes the morbid anatomy and symptoms, which are mainly those of indigestion. He concludes his account of these by saying, "a most important symptom, if present, is glycosuria, for the disease then is likely to put on the character of a severe diabetes." Both by this sentence and by the article as a whole, he implies that the disease may exist without diabetes. No doubt this is true, for the pancreatic change must have progressed considerably before the diabetes appears, but whether before this happens there are all the symptoms Fitz describes is, I think, very doubtful. Most diabetic patients do not give a history of these symptoms having preceded the onset of the diabetes. Naunyn gives no such symptoms as pointing to pancreatic diabetes. I think we may fairly conclude from our post-mortem experience at Guy's, that, apart from chronic pancreatitis due to the spread locally of inflammation from neighbouring organs, there are only two undoubted and important forms, one due to the venous engorgement, and the other associated with diabetes, and that there is no evidence that chronic pancreatitis produces any symptoms, except those of diabetes.

Leo gives a short description of atrophy of the pancreas, and mentions the frequent association with diabetes. He mentions arteriosclerosis as a cause, but of that we have no instance among the post-mortems at Guy's.

Lancereaux does not give a complete description of the conditions of pancreatic disease which cause diabetes, but he states that pancreatic disease will do so, and gives some cases of fatty pancreas in which diabetes was present.

Oser gives a very full account of the subject. He considers it proved that although one cause at least of diabetes is disease of the pancreas, yet this organ may be perfectly healthy, as far as the microscope can discover, in cases of diabetes, and also the pancreas may be totally destroyed by disease without there being any diabetes. It appears that pancreatic diabetes is nearly always associated with disease of the whole pancreas. Simple atrophy of the pancreas is by far the commonest disease of the pancreas associated with diabetes; for instance, Hausemann

analysed the cases from the Berlin Pathological Institute for ten years and found :—

Diabetes without pancreatic disease	8 cases.
„ „ mention of the pancreas ...	6	„
„ with disease of the pancreas ...	40	„
Pancreatic disease without diabetes ...	19	„

Among the forty cases of disease of the pancreas in which diabetes was found were :—

- 36 Simple atrophy.
- 3 Fibrous induration.
- 1 Complicated case.

We see, then, that at Berlin, as at Guy's, diabetes is commonly associated with simple atrophy of the pancreas, indeed far more commonly than any other disease of the pancreas. The proportion of cases of diabetic pancreatic atrophy to those of diabetes without pancreatic disease, and to those of pancreatic disease without diabetes, is higher at Berlin than at Guy's, but this is of small importance as it probably depends upon varying opinions as to whether a pancreas which is small should be considered as normal or slightly atrophied.

Hausemann thinks that diabetic and "cachectic" pancreatic atrophy can be distinguished. In the cachectic the surrounding fatty tissue is atrophied, the pancreas is somewhat cylindrical. Microscopically the condition is one of simple atrophy without any pigmentation. In the diabetic atrophy the pancreas is flabby and dark-coloured, especially its connective tissue ; it is flat and is more fixed by its surrounding fatty and connective tissue, some of which may be newly formed. Microscopically, there is no special change in the secretory cells, but the connective tissue has undergone changes which are like those of an atrophic granular kidney. He, indeed, regards this condition of pancreas as giving its own special symptoms, just as granular kidney gives its own. The cases I have collected do not enable us to say whether Hausemann's distinction is valid. The question can only be settled by very careful investigation of many post-mortems.

I have quoted from Oser only so far as is necessary to show that our experience at Guy's corresponds with his statements. Those who wish for fuller information on the subject of pancreatic diabetes cannot do better than consult his article. He shows that almost any disease of the pancreas may in rare instances be associated with diabetes. He gives references to cases in which it occurred with the following pancreatic diseases:—Fatty degeneration, indurative pancreatitis, pancreatic calculi (which may secondarily cause serious disease of the gland) carcinoma, cysts, abscesses, hæmorrhage, necrosis, and fat necrosis. Sometimes the disease was only detected on microscopical examination. We do not appear to have had at Guy's any examples of bronzed-diabetes in which the skin is bronzed and the patient has diabetes. At the post-mortem the liver is hard and brownish red and there is degeneration of the liver cells, increase of fibrous tissue, with a deposit of pigment, and the pancreas undergoes similar changes.

GROWTHS IN OTHER ORGANS ADHERENT TO PANCREAS

Thirteen of our cases were examples of this. Usually the growth is not only adherent to but invades the pancreas. Most often it is a carcinoma of the stomach, namely, in seven cases out of thirteen, and of these in five the growth was in the pylorus; naturally the head of the pancreas is the part to which the pyloric growth is usually adherent.

In four cases, malignant disease of the abdominal glands affected the pancreas, and in one of these the affection of the glands was secondary to that of the stomach. In one case a growth of the colon had extended to the tail of the pancreas, and in another a growth of the right kidney had affected the head. There is nothing to be gained by analysing these cases further, for it is clear that if the pancreas is sufficiently infiltrated by growth there will be present the symptoms of malignant disease of the pancreas together with those due to the primary growth. In such a case as 1896, 321 it would be easy, as jaundice was well marked, to consider the case to be an example of primary growth in the pancreas.

Our experience at Guy's confirms Lancereaux's statement that the stomach is the organ from which growth most frequently extends directly to the pancreas. The only other organs he mentions are intestine and liver, but our figures appear to show that abdominal glands are a more common source than intestine or liver for the invading growth.

SECONDARY GROWTHS IN THE PANCREAS.

Eleven of our cases fall under this heading. Four at least were cases of sarcoma, two of the mediastinum and two of bones. Of the seven other cases, certainly most, and perhaps all, were examples of carcinoma; the primary growth was in the rectum, sigmoid flexure, ovary, breast, peritoneum, orbit and œsophagus. Sometimes the growth in the pancreas was a solitary nodule, sometimes two or even several such were present, or it might be so large that it could hardly be called a nodule. The secondary deposits may be anywhere in the organ, but whenever present in the pancreas they are also to be found in many other organs of the body. I doubt whether secondary deposits in the pancreas are ever recognised during life, for if, as in 1886, 247, the secondary pancreatic deposit is very large, its recognition will probably be prevented by other large secondary masses around it. It may be difficult when other organs near the pancreas are affected with growth to be sure whether the pancreatic growth is a genuine secondary deposit or is the result of direct invasion.

Lancereaux does not add anything to the teaching of our cases; he, however, refers to an interesting case in which Ackeren found mallose and indican in the urine and striped muscle-fibre in the intestine of a man who had cancer of the stomach with secondary deposits in the pancreas. Oser states that secondary deposits in the pancreas are much more common than the primary growth in the organ, but as I have already said, even if we add our thirteen cases in which the growth simply extended into the organ to the eleven in which it was truly secondary, we only have twenty-four cases against thirty-one of primary growth. He points out how much more common it is for the primary seat to be in the stomach than in any other organ.

FATTY PANCREAS.

Nine examples of this occurred in our post-mortem room during the fourteen years under consideration. In four of them there was severe backward pressure from the heart or lungs, for a nutmegged or congested liver was present, and in a fifth, 1890, 36, the circulation was evidently failing, for the patient, an old man of 78, had a fatty heart, atheroma of the aorta, and much bronchitis. Therefore, increased backward venous pressure certainly appears to be a cause of fatty pancreas. Probably, too, a general wearing out of the tissues has also something to do with the production of this change in the pancreas, for of the five cases just mentioned three were over 70 years of age, namely, 72, 75, and 78. And of the remaining four cases that go to make up the nine, one succumbed to a fractured tibia and fibula; another, who is said to have looked very much older than his age, and showed a fatty liver, died after excision of the knee; the third, a woman aged 59, succumbed during an operation for strangulated hernia; and the fourth was a man who, at the age of 45, had atheroma and cerebral hæmorrhage. There appear to be all degrees of fatty degeneration up to the condition in which the whole of the pancreas, except the head, is said to be replaced by fat. It is not possible always to be sure whether the cases were instances of fatty infiltration or fatty degeneration of the pancreas, but 1888, 325; 1889, 331; 1891, 185 seem to be three instances of fatty degeneration, and 1885, 72; 1890, 36; 1892, 35; and 1897, 100 instances of fatty infiltration, but the numbers are too small, and microscopical examinations too few for it to be worth while to attempt to draw conclusions about the separate conditions from this series, especially as probably fatty infiltration may mix with fatty degeneration.

In none of the cases here collected did the report describe them as examples of fat necrosis.

Neither Leo nor Fitz say much about fatty pancreas. Lancereaux describes fatty infiltration as commonest in alcoholic subjects, and says it may be part of a general obesity. Fatty degeneration, too, he says, may follow alcoholic excess, but it may also be due to arteriosclerosis or obstruction of the pancreatic duct. He gives

some interesting cases of fatty pancreas in association with diabetes; there are quite enough cases on record of this association for it to be of undoubted importance, but we do not appear to have had a case at Guy's, although it may be that when patients die quickly after injuries or surgical operations diabetes has been overlooked.

Oser gives a short account of fatty degeneration. He gives as causes for it, infective diseases, retention of pancreatic secretion, alcoholic and phosphorus poisoning. It is interesting to note that during ten years' work in the post-mortem room at Vienna ten cases of fat necrosis of the pancreas were found, but only two of fatty degeneration. He gives references to many cases of fatty pancreas in association with diabetes, and also points out that fatty degeneration and fatty infiltration merge one into the other; indeed it is not clear under which heading some of the cases of diabetes should be placed.

Contrasting our experience at Guy's with that of others, it appears that we see a considerable number of instances of fatty pancreas, that as none of our cases showed cirrhosis of the liver, alcohol is not such an important factor as some authors think, but that they do not lay enough stress on backward pressure and general degenerative changes as a cause of fatty pancreas.

Fat necrosis on the surface of the pancreas occurred at Guy's in one case of rupture of that organ (see p. 60), and fat necrosis of the omentum was present in one case of malignant disease of the pancreas.

DILATATION OF THE PANCREATIC DUCT.

Four cases are entered under this heading in the table at the beginning of this paper, but there are also scattered under other headings, eight cases not entered in the preliminary table, because no case is there entered twice over. The whole twelve cases may be thus grouped according to the cause of the dilatation.

Malignant disease of the pancreas, 1887, 189;						
1892, 214; 1893, 137; 1896, 452	4
Calculi	3
Malignant growth of duodenum	2
Not obvious	2
Constriction at entrance to duodenum	1

In two of the cases of malignant growth of the pancreas, the ducts were not only dilated but cystic, some of the cysts being one inch across, and in one case the ducts were dilated, tortuous, and filled with colourless fluid. It is curious that malignant disease of the pancreas compresses and causes dilatation of the bile duct much more frequently than of the pancreatic duct. In all the cases in which dilatation of the pancreatic duct was due to pancreatic growth, this was primary in the pancreas. The three cases of calculi will be described under that heading. The obstruction to the duct that followed malignant disease of the duodenum, although in one case it caused the duct to be the size of a 12 catheter, does not seem to have caused any naked-eye structural changes in the pancreas. The two cases in which the cause of the dilatation was not obvious are 1892, 223, a man aged 56, who died of bronchitis with emphysema, and 1888, 112 (described under suppuration of the pancreas) a man aged 57, who died of carcinoma of the sigmoid flexure; there was a small abscess in the head of the pancreas and the duct was dilated. In both these cases the dilatation was considerable, in the last the duct would admit the finger. Possibly in both these cases the dilatation was due to a calculus which had been passed.

In 1888, 209, both pancreatic and biliary ducts were dilated, and as they were constricted at their entrance to the duodenum, it seems reasonable to suggest that the dilatation was due to constriction following the healing of a small duodenal ulcer.

Lancereaux gives an account of dilatation of the duct, dividing it into a general dilatation and a cystic dilatation, the last he considers rare. He remarks, that if the block is complete and long lasting, there will follow an accumulation of mucus in the duct, with increase of the fibrous tissue in the gland, and atrophy of it. Other authors do not give a separate description of dilatation of the duct.

TUBERCLE OF PANCREAS.

Four cases of this were found in the Guy's post-mortem room during the period from which our cases were collected. Three of the patients suffered from general tuberculosis, and one from tubercular peritonitis. The affection of the pancreas was never

severe, and indeed, in two at least of the cases it was only slight. Like tubercles elsewhere, pancreatic tubercles may caseate, but in none of our cases did they break down. Pancreatic tuberculosis is clearly an unimportant condition.

Leo says, that it is doubtful whether tubercle of the pancreas even forms a part of acute miliary tuberculosis, and he says, that it is seen as caseous foci, surrounded by tubercles. Some of our cases were in an earlier stage than this, for in one the pancreas only contained a solitary tubercle.

Lancereaux considers that tubercle of the pancreas is wrongly thought to be rare, and he quotes Kudrewetzki as finding that among a hundred and twenty-eight cases of tuberculosis, the pancreas to be affected fifteen times, five times as part of an acute miliary tuberculosis, seven times as part of a chronic tuberculosis, and twice from tuberculosis in its neighbourhood. He describes two forms, one characterized by the presence of large caseous masses, while in the other there are granular infiltrating tubercles. He mentions one case in which a caseous mass broke down into an abscess.

Oser gives many references to authors who consider pancreatic tuberculosis rare, but he quotes as the latest opinion, that of Kudrewetzki already referred to by Lancereaux. Oser states that 12 undoubted cases of tuberculosis of the pancreas were found in Kudrewetzki's series of cases of pancreatic disease, or 9·37 per cent. This author found 44·44 per cent. of cases of tubercular disease of the pancreas in tubercular children. Our experience at Guy's is certainly not that of Kudrewetzki, for with us tubercle of the pancreas is found in far under 1 per cent. of all cases of tuberculosis. Our experience, indeed, agrees with that of the majority of authors. Oser describes an extremely interesting case in which a tubercular tumour was excised from the pancreas.

MECHANICAL DAMAGE TO THE PANCREAS.

There are four examples of this in our series. Two of the patients were boys who were run over; in both many of the abdominal contents were torn, and in one the head of the pancreas was pulped and its duct torn across, and in the other, it was torn in its middle. The third patient was crushed, and there

was hæmorrhage around the pancreas and in its tail. The fourth was an extraordinary case; the patient was struck by a shaft of a cart; he lived a week. The great omentum was torn, there was a rent in the head of the pancreas and much fat necrosis in the omentum, mesentery, meso-colon, subperitoneal fat of the abdominal wall and on the surface of the pancreas. This case teaches that life may be prolonged for a week after a tear in the pancreas and appears to show that fat necrosis may attain an advanced stage in the same time.

Lancereaux states that the pancreas may be contused or torn across, in which case the diaphragm is sometimes ruptured and the pancreas may pass into the thorax, and he also states that if the patient live long enough the blood effused in the neighbourhood of the pancreas tends to form a cyst. Previous injury has often⁶ been suggested as a cause for certain obscure cysts found at the upper part of the abdomen, but it is noteworthy that in our patient, who lived a week, there was no formation of a blood cyst. Oser draws most of his information from Leith's paper⁷ and states that usually other organs are much damaged as we have found at Guy's. It appears highly probable that a severe wound of the pancreas is in itself not necessarily fatal, for our fourth case lived a week, Oser states that Cooper's case lived some days, that Wagstaff's case lived five days, that in Rose's case of abdominal injury, in which the abdomen was drained and the patient recovered, a fistula which remained discharged fluid having the character of pancreatic fluid. Lancereaux quotes cases which occurred during the American Civil War. In these a bullet had injured the pancreas, but death appeared to be due to other damage than this; he considers that some forms of pancreatic cyst are due to injury of this organ. It may be worth while, in an exploratory laparotomy for abdominal injury, to examine the peritoneal fluid to see if it gives the reactions of pancreatic fluid, for this examination may help us to discover if the pancreas is damaged.

⁶ See Fisher, Guy's Hosp. Rep. Vol. xlix., p. 275.

⁷ Ruptures of the Pancreas, Edin. Med. Journ., Nov., 1895.

ULCER OF STOMACH ADHERENT TO PANCREAS.

We have four instances of this condition. There are all degrees of it. The stomach may be simply adherent to the pancreas, or so much of the stomach may, after adhesion, have been ulcerated away that on opening the stomach from the front the pancreas and other structures at the back of the abdominal wall may be seen. I have known such a specimen to be exhibited as an instance of ante-mortem digestion.

CALCULI IN THE PANCREAS.

During fourteen years we have had three examples of these in the post-mortem room at Guy's, that is to say, they were found in three bodies out of 6,708. All were found in elderly males. These calculi, which were white, all resembled some renal calculi more than biliary, in that they were irregular in shape, jagged and even branched. Thus, in the first case they were very numerous, like white coral; in the second there were a large number of irregular white jagged calculi; and in the third there were very many small white branching calculi. In all cases they were numerous, in two the ducts were dilated, and in the third there was a small cyst which contained white mucoid fluid; in the first two the pancreas had become firm and hard, and in the first it had atrophied, and, as there was sugar in the urine, it is, I think, fair to assume that its presence was due to the condition of pancreas although the patient apparently did not die from diabetes; but remembering the frequency with which diabetes appears to be the result of atrophy of the pancreas, it is probable that had he lived long enough he would have had the usual symptoms of diabetes.

Fitz, like Lancereaux, thinks that retention of the pancreatic secretion may be a cause of calculi; but, as Oser points out, this cannot be the only cause, for the orifice of the duct may be blocked without any stones being found, and further, in none of the three cases here recorded was there any evidence that the duct was blocked. The fact that the stones contain a good deal of carbonate of lime, although there is none in the pancreatic secretion also shows that the cause is not mere retention. The cause of pancreatic stones is unknown.

It is curious that all three Guy's cases were over fifty, for according to Oser they are commonest in the decade from thirty-five to forty-five. Eleven out of thirty-two cases to which he refers occurred then. Lancereaux considers them most frequent between thirty-five and sixty years of age. All the three Guy's cases were males, and there is no doubt they are commoner in men than women. Oser gives the proportion of men to women as twenty-six to six.

As at Guy's, so elsewhere, pancreatic calculi are usually multiple; Lancereaux says, as many as three hundred have been found in the gland; they may be any shape, smooth or irregular, sometimes long, lying in the ducts. Oser mentions one two and a half inches long. They are usually white. It is quite common for them to be associated with dilatation of the ducts or even the formation of cysts; further, they frequently lead to a hard atrophied gland with much fibrous tissue in it. Indeed, Oser says, that as gall-stones lead to biliary cirrhosis, so pancreatic stones lead to pancreatic cirrhosis, but judging from the Guy's cases and from those described by authors, I think there is no doubt that pancreatic stones proportionately much more frequently lead to severe pancreatic atrophy than gall-stones lead to severe biliary cirrhosis.

Sometimes the ducts may be encrusted with deposit, and there may be pancreatic sand, comparable to biliary sand.

Fitz, Leo and Oser all state that calculi may be associated with cancer of the pancreas. They are, however, so rare that the association must be very uncommon. None of our cases of pancreatic carcinoma showed a stone and none of the three cases of pancreatic calculi showed cancer of the gland. Considering the frequency with which carcinoma of the gall-bladder is associated with gall-stones, there is nothing surprising in the fact that pancreatic stones are met with in pancreatic cancer. Lancereaux and Oser state that these calculi may also occur in those who are the subjects of renal or biliary calculi. Our cases do not illustrate this. Oser quotes Gindicandrea as finding two cases of pancreatic calculi among 122 post-mortems—or in 1·64 per cent. of cases. Our series suggests that this was exceptional, for out of 6,708 post-

mortem examinations at Guy's pancreatic calculi have only been found three times. It is a matter of great interest that the urine in one of our cases contained sugar. Oser gives an interesting table of twenty-two cases of diabetes associated with pancreatic stone, and it appears that, as was probably the case in our patient, the stone causes diabetes, because it leads to fibrosis and atrophy of the pancreas. Occasionally the gland is a little fatty. Lancereaux gives an example in which the presence of severe epigastric colic, glycosuria, and fatty stools led to a diagnosis of pancreatic calculus, but the stone was not found in the stools and there was no post-mortem. Oser, on the other hand, gives a case in which a pancreatic calculus was found in the stools, but there was no glycosuria. Nevertheless, there seems every probability that it is possible to diagnose the presence of pancreatic calculi, the chief symptom being severe epigastric colic, which for a diagnosis to be possible should be accompanied by one or more of the following symptoms: glycosuria, fatty stools, the presence of undigested muscle-fibres in the stools, and according to Lancereaux, in rare cases the presence of fat in the urine; this fat, he thinks, is absorbed from the intestine and is that which the pancreatic juice has failed to digest. The finding of a pancreatic calculus in the stools would confirm the diagnosis. It appears probable from cases recorded, that persons may successfully pass pancreatic calculi and get quite well. I need hardly add that some cases of pancreatic calculi probably produce no symptoms.

SUPPURATION IN THE PANCREAS.

Three examples of this occur in our series. In one, a patient who died of carcinoma of the sigmoid flexure had a small abscess in the head of the pancreas. The duct was much dilated and no cause could be found either for the abscess or the dilatation of the duct.

The second case was that of a young woman who suffered from general pyæmia secondary to a sloughing appendix. The pancreas contained numerous abscesses of considerable size, which were chiefly in the head. Pyæmic abscesses of the pancreas are excessively rare. According to Oser no case occurred in the post-mortem room at Vienna during ten years.

Our third case was one of acute general pancreatitis. I need not further allude to it, for it has been fully described by Dr. Newton Pitt in the *Clinical Society's Transactions*, 1898-99. It occurred in the last year of our series, 1897, and two other instances of acute pancreatitis occurred in 1898. Thus, in the two years, 1897 and 1898, we had three cases, and not one in the previous thirteen years, although the condition could hardly be overlooked. Several cases have been quite recently recorded by various authors. There is little doubt that acute pancreatitis is due to a micro-organism, and these facts suggest that lately we have been going through a period of unusual activity of this disease. There would be nothing surprising in this, for many diseases due to micro-organisms have periods of special activity.

So much has been recently written on the subject of acute pancreatitis, that nothing would be gained by our going over the subject with only one case as a basis for discussion.

Lancereaux refers to a case of abscess of the pancreas due to the pneumococcus, but we have no example of this.

PANCREATIC CYSTS.

Our series contains three instances of pancreatic cysts, in addition to the case already mentioned, in which calculi caused cystic dilatation of the duct, and a few cases in which a malignant growth in the pancreas was becoming slightly cystic. One of the three was an instance of that very rare condition in which cysts of obscure origin occur in many organs in the same patient. This patient was under Dr. Pye-Smith, and the case is recorded in the *Pathological Society's Transactions*, vol. xxxvi., where in vol. xxxiv. will be found somewhat similar cases recorded by Dr. Mahomed and by Dr. Savage and myself. They are fully discussed in these papers. Another of the three was a boy aged six, in whom a large hydatid of the head of the pancreas pressed upon the bile-duct, leading to dilatation of the bile-ducts and gall-bladder and cirrhosis of the liver. The patient presented all the common symptoms of retention of bile. Hydatids of the pancreas are excessively rare, so rare indeed that Lancereaux and Oser hardly refer to them.

Our third case is an example of the ill-understood condition that is commonly meant by the term cyst of the pancreas.

Lancereaux only describes cysts of the pancreas under the heading of their causes, such as adenomatous cysts, traumatic cysts, retention cysts, blood cysts, &c., so that as it is usually quite impossible to be sure of the origin of the cyst, his account is not available for comparisons.

Fitz, Leo and Oser all incline to the view that a contributing cause for these cysts is sometimes obstruction to the duct, and perhaps this view is supported by the fact that the cyst wall in our case contained calcareous material, suggesting that similar material had formerly blocked the duct; and this suggestion is supported by the history, for the patient had, four years before admission, suffered from attacks of colic. But, as Senn points out, obstruction to the duct cannot be the sole cause of these cysts, for there may be considerable obstruction without a cyst, and ligature of the duct in animals does not lead to the production of a cyst. Very often, as mentioned a few pages back, a blow seems to have some causative relationship to these cysts, but there was no history of injury in our patient. The cyst in our case was much larger than is usual, for it contained six pints of fluid; as is usually the case, its walls were fibrous, but it is exceptional for there to be no pancreatic tissue left, but as this was so in our case, we are unable to say in what part of the pancreas it arose. The fluid in it was thick yellowish red, and no doubt the red colour was due to blood, for it is common for the fluid to be of such a colour as to suggest the presence of altered or unaltered blood. It contained leucin and tyrosin; this, according to Oser, is rare. In our patient the stomach was in front of the cyst at the post-mortem and probably during life, for resonance could be obtained over the tumour. The stomach is often in front during life. Oser gives admirable diagrams showing that the cyst may either have the stomach in front of it, or pressing the stomach downwards appear between the liver and it, or pressing the stomach up appear between the stomach and colon, or pressing the colon forward have this and the stomach in front of it, or pressing the colon up appear below this. Our patient was unusually old,

namely, sixty-eight, for, according to Oser, sixty-two out of one hundred and twenty-one cases occurred between twenty and forty years of age and but few over sixty. Pancreatic cysts are equally common in men and women. Our patient had glycosuria—a most important symptom. Oser gives nine cases of pancreatic cyst with glycosuria. Our patient, as is commonly the case, had diarrhoea, and if this is associated with fatty stools and the presence of undigested muscle-fibres, the motions become a great aid to diagnosis. He complained of loss of flesh, and this is a common symptom. The tumour moved with respiration; Oser says this sometimes occurs. This is not surprising, for if it is large enough to press against the liver it will move with it.

HÆMORRHAGE INTO THE PANCREAS.

There are three instances of this in our series; in one the patient had had many fits, in another there was considerable venous backward pressure from heart disease, and in the third there was much in the lungs that might have caused great impediment to the venous flow. We may, I think, conclude that in all three cases the hæmorrhages were due to venous congestion. The blood was effused into the substance of the gland; in one case there were several hæmorrhages in it, in the others only one.

Lancereaux mentions backward venous pressure as a cause for venous congestion of the pancreas, and Oser refers to eclampsia and cardiac and pulmonary diseases as causes. In one year, in the post-mortem room of the General Hospital at Vienna, there were two cases, in one the hæmorrhage was associated with cardiac disease and in another with emphysema. Oser also refers to other cases. Leo speaks of hæmorrhages which not uncommonly result from venous obstruction in diseases of the circulatory apparatus, the lungs or the portal system, but I think he exaggerates their frequency. The striking thing to learn from our experience at Guy's, and from the literature of the subject (except Leo's article) is the rarity with which hæmorrhage is found in the pancreas although so many persons suffer from backward venous pressure from the heart, lungs, or liver, from Bright's disease, from blood diseases, or general embolism. It is curious that

emboli in the pancreas should be so rare, as they are so common in the spleen.

During the period named there has been no instance at Guy's of that form of pancreatic hæmorrhage in which the blood occupies the whole of the gland and the patient rapidly dies. Many instances of this disease have been recently recorded; probably we are passing through a period of unusual activity of hæmorrhagic pancreatitis.

LYMPHADENOMA OF THE PANCREAS.

One example of this has occurred at Guy's during fourteen years. The patient died from Hodgkin's disease, and the centre of the pancreas contained a lymphoid tumour. This is an excessively rare condition. Lancereaux is only able to give references to two cases and Oser does not refer to it.

The remaining cases in our series, namely, those illustrating a peculiar cavity in the head of the pancreas, an ulcer of the duodenum adherent to the pancreas, a kidney adherent to the pancreas, the action of acid on the pancreas, and an accessory pancreas, are sufficiently described in the appendix.

APPENDIX

Showing all the cases in which the Pancreas has been found in any way abnormal in the Post-mortem Room of Guy's Hospital during the fourteen years 1884-1897, both inclusive.

(The first number indicates the year in which the patient died ; the second the number in the Guy's Hospital Post-mortem Records.)

PRIMARY MALIGNANT DISEASE OF THE PANCREAS.

1886, 348. F., æt. 55. Dr. Pye-Smith.—Malignant disease of the head of the pancreas. The head of the gland was made up of hard, new growth, which extended almost to the middle of the organ. It yielded a juice on scraping. The liver was full of secondary nodules. The bile-duct could be traced through the growth and it was difficult to explain why it was not compressed. Eight months ago severe pain in stomach ; then much retching and brought up blood twice. On admission, tumour size of hen's egg seen in region of the head of the pancreas. It was very tender, great pain was the main feature of the case, and she died October 7th, 1886.

1886, 351. M., æt. 38. Dr. Goodhart.—Malignant disease of the head of the pancreas. The head of the organ was surrounded and involved in a soft fleshy-pink growth, which was the consolidated mass felt during life. The common bile-duct could not be traced into the duodenum, it appeared to end in the growth of the head of the pancreas. The gall-bladder was much distended with dark bile. Illness commenced early in July with sudden pain in pit of stomach. Has vomited several times. Jaundice nine days before admission on September 11th. Motions pale, bile in urine ; much vomiting. Liver a little enlarged, and from it a rounded protrusion projects. September 21st, passed blood in his stools. September 28th, a lump, two inches by two inches, can be felt below the xiphoid. He went steadily downhill, and died October 9th. No secondary deposits anywhere.

1886, 329. M., æt. 28. Dr. Goodhart.—Admitted September 3rd for tumour of abdomen. Pain (wearing) in abdomen one month before. On admission, very pale motions, liquid, contained blood. Lump felt, three inches by three and a half inches, deeply seated below and to left of umbilicus. September 11th. Vomiting brown matter ; vomiting increased, pain very great. Died exhausted on September 27th. Large retroperitoneal small-round-celled sarcoma of pancreas, involving duodenum. Pancreas wasted body and tail ; head deeply involved in growth, so that it could not be distinguished, tail contained a secondary growth. Ducts and gall-bladder normal. Secondary deposits in omentum, mesentery, kidneys and lungs.

1887, 189. M., æt. 54. Dr. Pye-Smith.—Cylindrical-celled carcinoma of pancreas, secondary growths in portal fissure, ascites, granular kidney. Eight months ago began to complain of pain, gradually got weaker and

emaciated; marked jaundice. On admission, very little abnormal beyond ascites. Post-mortem, large hard mass in head of pancreas, tail shrunken. Mass contained dilated ducts and cysts full of opalescent white material. Surrounded portal vein and bile-duct but did not grow into them. Many secondary nodules in the liver. Much jaundice, bile-ducts dilated and filled with mucus. Gall-bladder filled with colourless mucus but not dilated. Altered blood in stomach. Many of the nodules in the liver were becoming cystic. These nodules consisted of irregular ducts lined with large columnar cells.

1887, 192. F., æt 54. Dr. Hale White.—She was only in the hospital five days, and she had vomiting for some months with progressive emaciation and abdominal pain. No tumour palpable at any time. No jaundice. Large cubical mass of growth in the head of the pancreas, two and a half inches across; it invaded no large vessels or ducts, although in contact with superior mesenteric and hepatic arteries and portal vein and aorta. It was firm, pale cream-coloured, fibrous; exuding from pancreatic duct was opalescent fluid. Growth was in upper part of head of pancreas; duodenum adherent and very contracted. No secondary growths. It did not affect the liver or bile-ducts. The gall-bladder was much enlarged, six inches long; contents pale; no duct obstructed. All other organs healthy.

1887, 365. M., æt. 48. Dr. Hale White (*Path. Trans.*, 1888, p. 96).—Admitted October 28th. Pain in abdomen with constipation for four weeks. Bowels only open four or five times since. No mælena. Retching, no vomiting. Hard nodule, size of filbert, in abdominal wall above umbilicus. Abdomen distended. November, vomiting set in, could not be checked. Much pain. Death, November 15th. Peritoneum studded with nodules of growth following the obliterated hypogastric arteries and umbilical vein (felt during life); many in mesentery. Liver not affected. Cancerous nodule pressed on neck of gall-bladder, but did not obstruct it entirely, although somewhat. Gall-bladder full of bile. Common duct normal. Liver normal. Pancreas, head contained a mass of hard white growth size of small orange. Secondary deposits in lumbar and mesenteric glands. One of the semilunar ganglia was infiltrated.

1888, 365. M., æt. 42. Dr. Goodhart.—Pancreas huge mass of cancer, invading body and head, surrounding gland tissue extensively implicated. Secondary growths in liver, lung, pericardium, heart, kidneys, peritoneum. Ducts of liver dilated, gall-bladder very distended; common duct pressed in by the growth near the pancreas. Symptoms: pains in back and stomach. Tenderness in epigastrium, gall-bladder distended. Much jaundice, and gradual emaciation.

1889, 85. M., æt. 70. Dr. Pavy.—Admitted September 19th, 1888, for jaundice. Discharged. Admitted again January 1st, 1889, with enlarged liver, much emaciation and jaundice. Died comatose February 26th. Head of pancreas indurated and infiltrated with carcinoma; it had run along the biliary duct and occluded it at entrance to duodenum. It may have been primary in duct. Many secondary nodules in liver, which was deeply bile-stained. Gall-bladder distended five inches long. Cystic duct dilated to size of finger.

1889, 445. M., æt. 58. Dr. Pye-Smith.—Admitted November 23rd for intestinal obstruction. December 5th, lumbar colotomy. Death next day.

Not wasted, no jaundice. Head of pancreas seat of scirrhus growth size of orange, very hard and fibrous. It had extended to surrounding parts, and so compressed transverse colon as to cause intestinal obstruction. No secondary deposits in liver, gall-bladder or portal glands. Mesenteric glands and peritoneum affected.

1890, 492. F., æt. 50. Dr. Pye-Smith.—Three to seven months ago noticed motions whitish, then pain in back and constant vomiting. On admission, thin and deeply jaundiced, small swelling in region of gall-bladder; gradually sank, and died three weeks after, viz., December 29th. Gall-bladder moderately distended. Cystic, hepatic, and common bile-ducts considerably dilated. Head of pancreas harder and firmer than usual, and forming a mass which pressed on the common bile-duct. Secondary growths in the pleura.

1891, 334. M., æt. 73. Mr. Lane.—Admitted for tumour and pain in abdomen: he had an umbilical hernia, and as the bowels were constipated the abdomen was opened, but the hernia contained healthy bowel. He died two days after. Pancreas head occupied by a tough white growth the size of a Tangerine orange. Microscopically, it was scirrhus carcinoma. Small secondary deposits in liver and portal glands. No dilatation of gall-bladder or ducts. No jaundice. Gall-stones in gall-bladder.

1892, 246. M., æt., 59. Dr. Perry and Mr. Jacobson.—Admitted July 5th. Last two years pain in left hypochondriac region. Six months ago noticed motions small. Six weeks had constipation; enemata only bringing away small scybala. Four weeks ago abdomen distended and painful, with occasional vomiting. July 12th, operated on; descending colon opened: it was empty; death on 14th. On peritoneum many small secondary deposits; cylindrical carcinoma. There was scirrhus glandular carcinoma infiltrating the head of pancreas; by continuity it directly invaded the pylorus. Liver and mesenteric glands, secondary deposits. No intestinal obstruction or jaundice.

1892, 373. F., æt. 33. Dr. Pye-Smith.—Admitted August 13th. Last June, pain at umbilicus and felt a lump there. Middle of July, vomiting. On admission, firm globular swelling in the epigastrium bulging the ribs. Very jaundiced. October 10th, eight pints yellowish blood-stained fluid withdrawn from abdomen. Died October 22nd. Much emaciated. Large malignant growth size of foetal head in head of pancreas. Many secondary growths in liver and glands of portal fissure. Common duct pressed on, gall-bladder distended.

1892, 399. F., æt. 45. Dr. Goodhart.—Admitted August 8th for pain and swelling in abdomen of eight weeks' duration. Ascites. August 26th: tapped, many lumps felt in abdomen; later on, secondary deposits felt in abdominal wall; tapped twice more; died November 6th. Much wasted, no jaundice, legs œdematous. Pancreas replaced by medullary sphenoidal carcinoma; peritoneum, liver and glands very much affected.

1892, 214. M., æt. 42. Dr. Taylor.—Admitted April 21st for jaundice and large liver. Much pain, wasting, jaundice and vomiting. Head of pancreas much enlarged from growth, and pancreatic ducts much dilated and sacculated. Bile-ducts much dilated, gall-bladder not. No secondary growths.

1893, 137. F., æt. 48. Dr. Goodhart.—Admitted February 17th. Jaundice for four months; gradually died from exhaustion on April 13th. Pancreas enlarged into a mass of carcinomatous growth, three inches in diameter; it pressed on common bile-duct; gall-bladder very much distended; numerous nodules of secondary growths in liver. The duct of pancreas dilated and cystic, in places one inch across. Secondary growths in lung, bronchial, mediastinal and aortic glands.

1893, 507. M., æt. 38. Dr. Pitt.—Admitted December 22nd for dyspnoea, cough, and epigastric pain. Much dyspnoea. Liver enlarged. Right abdominal muscles rigid. Died dyspnoeic 29th December. No jaundice. Large mass of carcinomatous growth nine by two by two and a half inches in head of pancreas. Pancreatic and biliary ducts not dilated nor gall-bladder, but much growth in portal glands, and secondary deposits in liver, mediastinum, lungs, and suprarenals. Much fat necrosis of omentum.

1894, 99. M., æt. 48. Mr. Lane.—Admitted 10th March. Had been ill only three months with pain in back. Last month, jaundice; also pain in region of gall-bladder with vomiting. Operation: no gall-stones found; sank in two days. Hard scirrhus growth in tail of pancreas; head free; neighbouring glands affected; secondary deposits in liver.

1894, 332. M., æt. 37. Dr. Pitt.—History very imperfect; he was wasted but not jaundiced. There was a large mass of growth occupying most of the pancreas, with secondary growths in liver, lungs, bronchial glands.

1895, 39. M., æt. 46. Dr. Hale White.—Primary carcinoma of pancreas, secondary growths in liver, lymphatic glands and lungs. Admitted January 26th, 1895, for enlarged liver, wasting, and pain on right side of abdomen. Looked very ill, was deeply jaundiced, edge of liver felt at umbilicus with nodules on its surface and rub over it. Dulness at base of right lung, râles on left side. The history was so short as to suggest primary malignant disease of liver. Jaundice increased, he became convulsed, and died February 1st, 1895. The pancreas weighed seven ounces and contained at its head a mass of growth the size of a fives ball, in its centre were one or two small cysts. Normal pancreatic tissue appeared to shade off gradually into the growth. The glands in the neighbourhood contained secondary growth. Liver weighed twenty pounds and was full of masses of secondary growth. The gall-bladder contained gall-stones. The common bile duct was dilated.

1895, 204. F., æt. 36. Dr. Shaw.—Primary carcinoma of pancreas adherent to duodenum. She was admitted June 4th, 1895, for profound anæmia, vomiting and abdominal tumour. First began to be ill six months before. She gradually lost strength and died. There was a mass of growth in head of pancreas. This was adherent to duodenum, the first two inches and a half of which were destroyed by growth, and connected with the duodenum was a cavity, formed by growth of glands, which had ulcerated. The mass of glands infiltrated with growth was readily palpable through the abdominal wall. Otherwise no secondary deposits. Histologically the growth was a rapidly growing carcinoma with little stroma.

1895, 494. M., æt. 69. Dr. Pye-Smith.—Admitted December 11th for abdominal swelling. For eight weeks has been getting weak, becoming jaundiced, and his feet have swollen. He was tapped, and after bile-stained ascitic fluid was drawn off, it was found that the liver reached down to the umbilicus, and was hard and nodular. He died December 13th. The primary

growth was a carcinoma of head of pancreas, which was much enlarged, and this growth caused a projection into the common bile-duct. Portal vein pressed upon by pancreatic growth. Liver, many secondary deposits; bile-ducts much dilated. Abdominal, thoracic and cervical glands full of growth. Pericardium adherent. Liver contained a calcified hydatid.

1896, 37. M., æt. 68. Dr. Goodhart.—Admitted January 18th, 1896. First felt abdominal pain in December, 1895. Took to his bed January 6th, 1896. From then the abdomen began to distend. January 20th. Twelve pints of straw-coloured fluid withdrawn. He died on February 1st from respiratory failure. Weight, nine stone four pounds. Permission obtained only to open abdomen. Head of pancreas normal. Body and tail infiltrated with dense malignant growth. Innumerable small growths all over peritoneum; the cavity contained seven pints of clear fluid. All other abdominal organs normal.

1896, 38. M., æt. 60. Dr. Goodhart.—Admitted December 10th, 1895, for jaundice. Four months before admission noticed jaundice, with occasional attacks of abdominal pain. On admission, general jaundice, much wasting but little pain; liver, enlarged and irregular, extends to one inch below umbilicus. January, 1896, pain very severe, and patient sank and died February 4th, 1896. There was a large firm mass of growth in the head of the pancreas, extending for a third of its length, surrounding tissue and bowels firmly adherent to it. It had ulcerated into the duodenum, and in so doing had laid open a vessel, bleeding from which had killed the patient. The bile-duct was completely occluded by growth, and ducts behind were much dilated. Several glands in neighbourhood were secondarily affected. The liver was very full of secondary nodules. There was nearly a pint of blood in the intestine. The stomach contained nearly half a pint. Microscopically the growth was a typical carcinoma.

1896, 51. M., æt. 69. Dr. Pye-Smith.—Admitted January 20th, 1896, for an enlarged liver which reached two inches below the umbilicus. The enlargement is said to have been noticed in September, 1894. He died February 15th, 1896. Weight, eight stone eleven pounds. The head of the pancreas was converted into a mass of growth three inches across and one inch thick, which had commenced to caseate. There were several secondary nodules in Douglas's pouch. The liver weighed two hundred ounces and was very full of secondary growths. The pancreatic growth had not compressed the bile-duct, hence no jaundice. A few glands near the pancreas were affected.

1896, 86. M., æt. 53. Dr. Taylor.—Noticed some swelling of abdomen in August, 1895; soon pain came on. Admitted January 13th, 1896. Severe pain. Liver reached below umbilicus and two hard masses could be felt on it. Feet and ankles soon swelled, next jaundice and ascites appeared; he wasted and died March 13th. Liver weighed about fourteen pounds. Pancreas contained a small globular mass of growth which projected from the upper margin near the centre, about two and a half inches in diameter. Liver was crammed with secondary nodules and there were some on the under surface of diaphragm. Five pints of fluid in abdomen.

1896, 196. M., æt. 52. Dr. Goodhart.—Admitted February 26th, 1896. Liver large, no nodules were felt on it. March 14th, ascites noticed. April

18th, paracentesis ten pints. May 3rd, nine pints. Gradually got weaker and died May 30th, 1896. Very jaundiced. Pancreas contained a growth, with secondary deposits in liver.

1896, 287. M., æt. 43. Dr. Hale White.—For three months before admission irregularity of bowels, abdominal pain and wasting. On admission, June 1st, liver much below ribs. An anæsthetic failed to reveal any abdominal tumour. July 4th, several nodules felt in abdominal wall between umbilicus and ribs. The patient continued ill. Malignant peritonitis was diagnosed. There were no fatty stools. He died July 15th, very wasted. There was a scirrhus carcinoma in the tail of the pancreas; the rest of the gland was free. There were secondary nodules in liver. Bile-duct not obstructed. A few glands near the pancreas were affected secondarily.

1896, 297. M., æt. 54. Dr. Hale White.—Came in July 10th, 1896, for hæmatemesis; no cause could be discovered. July 14th, another attack. 18th, another with melæna. Died July 20th, very anæmic. In the central part of the pancreas was a very hard mass of growth which proved to be a glandular carcinoma. Its anterior surface was firmly adherent to posterior surface of stomach near lesser curvature and raising the gastric wall. The adherent wall of the stomach was infiltrated with growth, which had in the centre denuded the whole of the stomach wall so that there was a circular gastric ulcer about as large as a threepenny-piece. This ulcer led into a cavity in the pancreatic growth the size of a walnut, which was full of blood-clot, but the bleeding vessel could not be found. The stomach and small intestines were both full of blood-clot. The growth was confined to the centre of the pancreas. There were no secondary nodules anywhere except one or two in the wall of the stomach near the growth.

1896, 452. F., æt. 47. Dr. Taylor.—Admitted August 17th for jaundice and hepatic pain of four months' duration. Gall-bladder distended. Operation. Two stones removed from common duct. Jaundice diminished a little. Fistula discharged bile, patient steadily wasted and lost ground, râles developed in lungs, and she died emaciated on November 5th. There was a dense columnar scirrhus growth in pancreas, an inch and a half long and an inch in diameter. The growth invaded the pancreatic and hepatic ducts. The pancreatic duct was dilated, tortuous, and filled with a colourless fluid. No scar could be seen where the gall-stones had been removed from the hepatic duct. Liver, no secondary growths. There were a few in the glands, near the primary growth, and two small ones in the pancreas, also some on upper surface of diaphragm and lungs.

1897, 220. M., æt. 57. Dr. Pye-Smith.—About Christmas, 1896, constant severe pain on right side of abdomen, and jaundice. He lost weight. On admission, signs of ascites. May 10th. Abdomen tapped; sixteen pints of chylous fluid drawn off. No tumour could be felt. May 18th. Similar fluid from left chest; ten ounces. He died on June 4th, apparently from vomiting. The head of the pancreas was enlarged, and contained a firm white fibrous growth. There were numerous small nodules on the peritoneum. Many glands contained secondary deposits. It was not clear which growth had pressed so as to cause chylous ascites. Stomach contained much sub-mucous blood extravasation. Liver normal.

CIRRHOTIC, CONGESTED OR HARD PANCREAS.

1885, 299. F., æt. 45. Dr. Moxon.—Early tubal and interstitial nephritis, dilated heart, thickened mitral valve, pulmonary apoplexy, nutmeg liver. Pancreas congested, with increase of fibrous tissue and thinning of secretory cells.

1885, 355. M., æt. 35. Dr. Moxon.—Aortic disease, nutmeg liver, hard spleen. Pancreas very hard.

1887, 60. M., æt. 30. Dr. Goodhart.—Fibroid disease of heart with hypertrophy and dilatation. Pancreas firm and hard.

1887, 251. F., æt. 13. Dr. Hale White.—Mitral incompetence. Viscera tough. Pancreas tough.

1887, 343. F., æt. 53. Dr. Hale White.—Hobnailed cirrhosis of liver which weighed thirty-four ounces. Ascites. Pancreas extremely tough, dense and hard.

1888, 7. M., æt. 65. Dr. Taylor.—Adherent pericardium. Cirrhotic, fatty nutmegged liver. Bronchitis. Pancreas tough, with microscopically some fatty change and increase of fibrous tissue.

1888, 365. M., æt. 52. Dr. Pye-Smith.—Old phthisis. Scarred, syphilitic, cirrhotic, fatty liver; pleuritic effusion. Pancreas very tough and hard. Microscopically much increased fibrous tissue which had in some places extended into the lobes; much increase of small cells.

1889, 203. M., æt. 69. Mr. Howse.—Diabetes, coma, sloughing ulcer of left foot. Pancreas large and much indurated.

1899, 251. F., æt. 53. Dr. Goodhart.—Bronchitis, emphysema dilated and hypertrophied heart, pleural effusion. Pancreas hard.

1890, 49. F., æt. 39. Dr. Pavy.—Phthisis, cirrhosis of liver, tubercle of peritoneum, ascites. Pancreas extremely tough and large.

1890, 345. F., æt. 28. Dr. Perry.—Mitral stenosis. Hard viscera. Nutmegged and cirrhotic liver. Pancreas œdematous.

1891, 99. F., æt. 28. Dr. Horrocks.—Ovariectomy done shortly after parturition. Very large ovarian cyst. Pancreas large and extremely indurated.

1891, 251. F., æt. 40. Dr. Pye-Smith.—Mitral incompetence with stenosis. Liver congested. Pancreas large and tough.

1891, 408. F., æt. 17. Dr. Hale White.—Mitral regurgitation. Nutmegged liver. Pancreas rather hard.

1892, 7. F., æt. 29. Dr. Pye-Smith.—Mitral and aortic incompetence. Lobar pneumonia. Pancreas very hard.

1892, 182. M., æt. 58. Dr. Pye-Smith.—Mitral stenosis. Tricuspid incompetence. Cirrhotic liver. Granular kidneys. Pancreas tough.

1892, 415. F., æt. 46. Mitral stenosis. Malignant endocarditis of aortic valves. Nutmegged liver. Pancreas very hard, feeling like cartilage.

1895, 36. F., æt. 14. Dr. Hale White.—Adherent pericardium. Aortic and mitral incompetence. General œdema. Nutmegged liver. Hard pancreas.

1895, 464. M., æt. 26. Dr. Goodhart.—Phthisis. Tuberculosis of ileum. Lardaceous spleen. Pancreas tough.

1896, 363. M., æt. 54. Dr. Perry.—Aortic regurgitation with backward pressure signs as shown by œdema of feet and hard kidneys. Pancreas much redder than normal; all the veins appeared dilated.

1897, 379. M., æt. 45. Dr. Pye-Smith.—Mitral and tricuspid regurgitation. Mitral stenosis, ascites, nutmeg liver, very hard pancreas. Hard spleen.

1897, 388. M., æt. 26. Dr. Taylor.—Mitral regurgitation, hypertrophied and dilated heart, nutmeg liver, congested stomach, pancreas very red, hard spleen, hard kidneys.

1897, 397. M., æt. 55. Dr. Hale White.—Primary carcinoma of liver, starting in bile-ducts. Thrombosis of portal vein and its branches. Pancreas very hard.

1897, 419. M., æt. 37. Dr. Pye-Smith.—Very extensive pulmonary phthisis; pancreas hard; stomach congested.

1897, 423. F., æt. 15. Dr. Hale White.—Mitral and tricuspid stenosis and regurgitation; nutmeg liver; congested stomach; hard pancreas.

1897, 456. M., æt. 26. Dr. Goodhart.—Mitral stenosis; hæmorrhage into stomach; hard pancreas; hard spleen; hard kidneys.

SMALL ATROPHIC PANCREAS.

1885, 152. M., æt. 30. Dr. Pavy.—Diabetes, coma. Pancreas decidedly small and ribbon like.

1885, 194. F., æt. 38. Dr. Taylor.—Diabetes, pneumonia. Pancreas soft flaccid and small.

1885, 230. F., æt. —. Mr. Brailey.—Diabetes, cataract. Pancreas decidedly small; only weighed one ounce.

1886, 192. F., æt. 46. Dr. Hale White.—Sarcoma of both ovaries, lumbar and mediastinal glands. Pancreas much pressed upon by glands and wasted.

1888, 113. F., æt. 29. Dr. Pye-Smith.—Diabetes. Pancreas distinctly small, soft and flabby; only weighed one and a half ounces.

1888, 150. F., æt. —. Dr. Pavy.—Diabetes; sloughing gall-bladder full of gall-stones. Pancreas hard, indurated, small.

1890, 17. M., æt. 32. Dr. Taylor.—Diabetes and phthisis. Pancreas perhaps rather small but it looked normal.

1890, 39. M., æt. 66. Mr. Brailey.—Diabetes. The pancreas seemed rather smaller than normal. It was flabby.

1890, 98. M., æt. 34. Dr. Shaw.—Diabetes. Pancreas certainly smaller than usual.

1890, 173. M., æt. 63. Mr. Davies-Colley.—Diabetes. Pancreas certainly smaller than normal,

1890, 229. F., æt. 56. Mr. Higgins.—Diabetes. Pancreas small and fibrous.

1891, 5. M., æt. 32. Dr. Pye-Smith.—Diabetes. Pancreas small and flabby.

1891, 26. M., æt. 62. Dr. Taylor.—Carcinoma of kidney. Pancreas not pressed upon, but only half its normal size. There is much general wasting and no fat in omentum.

1891, 88. F., æt. 58. Dr. Goodhart.—Diabetes, coma. Pancreas decidedly small and flabby.

1892, 44. F., æt. 76. Mr. Durham.—Severe burn, died in less than forty-eight hours, delirious. Pancreas extremely atrophied and small, its body did not measure half an inch transversely.

1892, 76. F., æt. 22. Dr. Goodhart.—Diabetes. Pancreas small and flabby.

1893, 321. M., æt. 36. Dr. Moxon.—Diabetes. Pancreas small, very dense and fibroid. Cutting like a cirrhotic liver.

1894, 358. M., æt. 45. Dr. Hale White.—Diabetes, coma. Pancreas very firm and fibrous; perhaps rather tough

1895, 56. M., æt. 64. Dr. Washbourn.—Diabetes and phthisis. Death from coma. Pancreas very small and hard. Weight one and a half ounces.

MALIGNANT GROWTH ADHERENT TO THE PANCREAS.

1885, 346. M. Dr. Carrington.—Cancer of pylorus attached to heart not invading pancreas.

1888, 11. M., æt. 63. Dr. Pye-Smith.—Cancer of pylorus, liver, mesenteric glands and suprarenal glands. The pyloric cancer was adherent to and infiltrating the pancreas.

1890, 58. F., æt. 42. Dr. Taylor.—Carcinoma of pylorus extending along the lesser curvature and involving the tail of pancreas.

1890, 353. M., æt. 43. Dr. Perry.—Carcinoma of pylorus involving head of pancreas.

1890, 189. M., æt. 47. Dr. Goodhart.—Carcinoma of lesser curvature of stomach involving pancreas.

1891, 396. M., æt. 46. Dr. Taylor.—Sarcoma of abdominal glands extending into head of pancreas.

1892, 5. M., æt. 59. Dr. Taylor.—Carcinoma of colon, growth adherent to tail of pancreas.

1892, 26. F., æt. 29. Dr. Pye-Smith.—Carcinoma of stomach, of liver, and the lymphatic glands of the portal fissure. Pancreas very hard and was invaded by growth.

1892, 101. M., æt. 59. Dr. Pye-Smith.—Carcinoma of stomach. The glands secondarily affected had invaded the pancreas, which was infiltrated with growth.

1896, 321. M., æt. 49. Dr. Pye-Smith.—Admitted for abdominal pain and jaundice. Enlargements could be felt on, and a mass below, the liver. Patient gradually sank and died. There was a large mass of carcinoma of the pylorus which had invaded the head of the pancreas and pressed on the common bile-duct.

1897, 28. M., æt. 54. Dr. Pitt.—Small-celled sarcoma of the right kidney, which was a large tumour and had infiltrated the duodenum and head of the pancreas. No jaundice.

1897, 71. M., æt. 47. Dr. Taylor.—Malignant disease of œsophagus. Secondary deposits in aortic and abdominal glands. The latter proved to be a mass weighing fifty-seven ounces, behind the stomach; it invaded the pancreas and both suprarenals.

1897, 134. M., æt. 49. Dr. Hale White.—Carcinoma of the stomach with secondary deposits in glands, and one of these glands had grown into the tail of the pancreas.

SECONDARY GROWTHS IN PANCREAS.

1885, 284. M., æt. 22. Dr. Pye-Smith.—Sarcoma of anterior mediastinum, secondary growths in right adrenal, pericardium, pleuræ, kidneys, lumbar and mesenteric glands. Pancreas contains a mass in the upper part of its head.

1885, 306. F., æt. 38. Dr. Taylor.—Lympho-sarcoma of superior mediastinum. Secondary growths in glands and ovary. Pancreas, one small hard secondary growth the size of a pea.

1885, 404. M., æt. 46. Mr. Lucas.—Cancer of rectum and bladder. Secondary growths in liver, suprarenals, glands. Pancreas was infiltrated with growth, especially the head.

1886, 78. F., æt. 1. Mr. Howse.—Multiple sarcomata of skull, ribs, liver. Tail of pancreas had a secondary nodule in it.

1886, 247 M., æt. 48. Dr. Goodhart.—Primary sloughing growth of sigmoid. Secondary disease of liver and retroperitoneal glands. On admission, August 9th, tumour felt two inches below left ribs, with a pulsation transmitted from aorta. Tumour grew, and patient sank and died on July 25th. Much diarrhoea from growth in sigmoid. There was a large hemispherical mass, measuring three inches either way and one and a half thick, in the abdominal wall. It was situated three inches above the umbilicus, over the cancerous head of pancreas. There was a large mass occupying the head of the pancreas, three inches in diameter. It had pushed the stomach over to the left, so that the first and second portions of duodenum ran below it. The liver was twisted round to the right. The mass consisted of cancerous head of pancreas and glands. The liver contained a few secondary growths. The left lobe much atrophied from pressure between pancreas and abdominal wall. No dilatation of ducts or gall-bladder. Right suprarenal completely atrophied.

1889, 68. F., æt. 36. Dr. Galabin.—Medullary carcinoma of ovary. Growths in bronchial, mediastinal, mesenteric glands, liver, spleen, suprarenals, and brain. Pancreas had a firm growth in head and tail while the intervening portion was free.

1890, 460. F., æt. 32. Dr. Hale White.—Carcinoma of both breasts. Secondary deposits in liver, lungs, gall-bladder and pleura. The pancreas was stony, hard, and under the microscope was seen to contain much spheroidal-celled secondary deposit.

1891, 238. M., æt. 50. Dr. Shaw.—Spheroidal carcinoma of peritoneum, liver, mesenteric glands, and tail of pancreas.

1891, 449. F., æt. 50. Dr. Taylor.—Spindle-celled sarcoma of ilium. Secondary deposits in liver and lungs. Pancreas contained several isolated nodules of secondary deposit.

1892, 449. F., æt. 57. Mr. Durham.—Malignant disease of orbit; secondary deposits in liver, lung, glands, and one small one in pancreas.

1895, 392. M., æt. 61. Dr. Perry. Carcinoma of œsophagus and stomach, with secondary growths in brain, pleura, lungs, liver, suprarenals, mediastinal and cervical glands; also head of pancreas contained a secondary growth about three-quarters of an inch in diameter.

FATTY PANCREAS.

1885, 72. F., æt. 16. Dr. Pavy.—Chorea. Acute endocarditis. Acute nephritis. Nutmeg liver. Pancreas infiltrated with fatty particles.

1885, 92. M., æt. 7. Dr. Pavy.—Thickened mitral valve. Dilated and hypertrophied heart. Nutmeg liver. Pancreas studded with minute yellow patches.

1888, 325. M., æt. 72. Mr. Howse.—Old ascending nephritis, acute pyelonephritis with suppuration, adherent pleura. Emphysema. Nutmegged

fatty liver. Pancreas, extreme fatty degeneration and infiltration; showed well with osmic acid.

1889, 331. M., æt. 75. Dr. Hale White.—Granular kidneys. Hypertrophied heart. Œdema of glottis. Liver congested. Pancreas with osmic acid showed much increase of fat in cells.

1890, 36. M., æt. 78. Mr. Davies-Colley.—Fractured femur. Bronchitis. Atheroma of aorta. Fatty heart. Cysts in kidneys. Pancreas small and infiltrated with fat.

1891, 30. M., æt. 51. Mr. Durham.—Fracture of tibia and fibula. Cellulitis of leg. Pancreas showed fatty patches in it.

1891, 185. M., æt. 48 (looked very much older). Mr. Jacobson.—Excision of knee, amputation of thigh. Liver very fatty. Pancreas, all except the head, might be said to be replaced by fat.

1892, 35. F., æt. 59. Mr. Lucas.—Strangulated inguinal hernia, gangrenous bowel, herniotomy, excision of gangrenous part, death during operation. Pancreas surrounded and partly infiltrated by fat.

1897, 100. M., æt. 45. Dr. Goodhart.—Atheroma, cerebral hæmorrhage. Pancreas normal except that it was infiltrated with fat.

DILATED PANCREATIC DUCT.

1888, 209. M., æt. 50. Mr. Symonds.—On March 27th, cholecystotomy for jaundice; no gall-stones found. May 27th. Gall-bladder and small intestine stitched together; fæcal fistula formed. Died June 18th. Abscesses in liver. Hepatic and pancreatic ducts much dilated; secondary to constriction at entrance to duodenum; no cause found.

1892, 223. M., æt. 56. Dr. Goodhart.—Bronchitis, emphysema. Dilated right side of heart. Pancreatic duct much dilated, otherwise normal.

1895, 141. M., æt. 41. Dr. Hale White.—This patient had had jaundice; much abdominal pain and ascites, for which repeated tapplings were necessary. The post-mortem examination revealed carcinoma of duodenum, which pressed upon the orifices of the bile and pancreatic ducts, which were so dilated that they would take a No. 12 catheter. There were growths in the peritoneum and glands. Thrombosis of hepatic and superior mesenteric veins and membranous colitis.

1895, 341. F., æt. 65. Dr. Goodhart.—In this case there was a cancerous ulcer of duodenum at the orifice of the pancreatic and bile ducts. The pancreas was healthy; there was jaundice.

TUBERCLE OF PANCREAS.

1885, 189. M., æt. 35. Dr. Taylor.—Tubercular peritonitis. Pancreas infiltrated in its connective tissue by caseous material, either in masses or as solitary tubercles. The glandular substance looked healthy, although the lobules were so surrounded.

1892, 152. F., æt. 19. Dr. Galabin.—Parovarian cyst with general tuberculosis. In the pancreas were several distinct caseous nodules about one-eighth of an inch in diameter, which were tubercular but did not arise from neighbouring caseous glands.

1896, 149. M., æt. 2. Dr. Washbourn.—This was a case of general tuberculosis in which there were a few small yellow tubercles in pancreas. No tuberculous peritonitis.

1897, 218. F., æt. 4½. Dr. Shaw.—A case of acute general tuberculosis in which one tubercle was found in the pancreas.

MECHANICAL DAMAGE TO THE PANCREAS.

1886, 315. M., æt. 16. Mr. Durham.—Run over; ruptured liver and duodenum; pulped head of pancreas and common bile-duct torn across.

1895, 379. M., æt. 9. Mr. Davies-Colley.—Cart-wheel went over his abdomen. Stomach, duodenum, kidneys, and spleen were ruptured, and the pancreas was torn across through the middle. The abdominal wall was injured.

1896, 183. M., æt. 12. Mr. Lucas.—Patient was crushed between the wheels of some machinery. The spleen was ruptured. There were twenty ounces of blood in the peritoneal cavity. There was hæmorrhage around the pancreas and in its tail.

1896, 329. M., æt. 53. Mr. Howse.—A cart-shaft struck patient on the right side of the abdomen. He lived a week. There was a mediastinal dermoid cyst. There was a rent in the muscles and peritoneum of the abdominal wall, and to the rent the great omentum had become recently adherent; the abdomen contained some blood-stained fluid. There was much fat necrosis in omentum, mesentery and meso-co'on, on surface of pancreas, and in subperitoneal fat of abdominal wall. There was an irregular rent in the duodenal end of the pancreas.

ULCER OF STOMACH ADHERENT TO PANCREAS.

1894, 300. F., æt. 67. Sir Samuel Wilks.—Ulcer of stomach, perforation. It was a very large ulcer and had chiefly for its floor the pancreas, which was indurated and hard, white on section, reminding one of growth; there were several bands of fibrous tissue extending into it.

1887, 310. M., æt. 39. Dr. Pye-Smith.—An old gastric ulcer was adherent to the pancreas, which was otherwise normal.

1891, 292. F., æt. 35. Dr. Pitt.—Cerebellar softening. There was a large gastric ulcer which had pancreas for its floor.

1895, 124. F., æt. 18. Dr. Pye-Smith.—Died of phthisis and pneumothorax. At the autopsy there was a chronic gastric ulcer which had eaten through all the coats of the stomach, and the pancreas formed its floor.

CALCULI IN THE PANCREAS.

1889. M., æt. 70. Dr. Goodhart.—Emphysema, bronchitis. Heart hypertrophied and dilated. Atheroma. Fibroid testes. Admitted 13th February, 1889. Was observed to have eight grains of sugar to the ounce. The glycosuria persisted till he died, but all his symptoms were referable to the condition of heart and lungs. Pancreas, a calculus was lodged in the duct three-quarters of an inch from the duodenal opening; duct dilated to size of a cedar pencil; very numerous calculi like white coral; head very fibroid; organ small.

1889. M., æt. 58. Dr. Pye-Smith.—Tuberculosis of lungs. Cancerous suppurating mesenteric glands with suppurative peritonitis. Pancreas firm and hard and contained a large number of irregular white, jagged calculi, and some of the ducts were dilated.

1892, 28. M., æt. 55. Dr. Goodhart.—Bronchitis, fibroid pleura and lung. Dilated and hypertrophied right heart. Pancreas contained very many small branching white calculi which were massed in the head in which was a small cyst about the size of a cherry, which contained white mucoid fluid. There was no suppuration.

SUPPURATION IN PANCREAS.

1888, 112. M., *æt.* 57. Mr. Durham.—Carcinoma of sigmoid. Pancreas thus described: In the connective tissue of the head was a small abscess; the duct was dilated so as to admit the finger. The cause of the dilatation was not obvious.

1895, 380. F., *æt.* 21. Dr. Newton Pitt.—Sloughing appendix with local abscess; operated on. Later, empyema of right chest, which was opened; it led to hepatic abscess. Patient sank. There were found, all secondary to the trouble about the appendix, suppurative pyelophlebitis, general suppurative peritonitis, pyæmic abscess of liver, empyema, and the pancreas contained numerous abscesses of considerable size, chiefly near the head.

1897, 447. Suppurative pancreatitis recorded in Clin. Soc. Trans., 1898-99. Dr. Newton Pitt.—It is noteworthy that this was the first case of acute pancreatitis in my series, but there were two more in 1898.

PANCREATIC CYSTS.

1884, 127. M., *æt.* 27. Dr. Mahomed.—Cyst in cerebellum and cysts in kidney and pancreas. The tissue of the pancreas seemed healthy. Small thin-walled cysts varying in size from a hemp seed to a hazel nut were scattered about here and there, some projected on surface; they were mostly in tail. There were about eight or nine in all. Fluid in them quite clear.

1894, 412. M., *æt.* 68. Dr. Goodhart.—Admitted 15th October for tumour in upper part of abdomen. Four years ago attacks of colic and vomiting. No history of injury. First three weeks, diarrhoea and loss of flesh. The swelling in the abdomen has only been noticed a few weeks. On admission, it is found to extend from the lower end of sternum to umbilicus, and four or five inches transversely, and adherent to liver. Showed non-expansile pulsation and was resonant. The spleen was a little enlarged. The urine contained five and a half pints per 1000 of sugar. On 16th Mr. Lane operated and found a cyst containing six pints of thick yellowish red fluid, unconnected with liver. The fluid contained leucin and tyrosin. Died on 24th, from heart failure. Not emaciated, no jaundice. The cyst was behind the peritoneum. The stomach was in front of it. It was in the situation of the pancreas. It had fibrous walls, which were thick and contained calcareous material, but no pancreatic tissue could be found. Spleen a little enlarged. Kidneys slightly granular.

HYDATID OF THE PANCREAS.

1894, 489. M., *æt.* 6. Dr. Pye-Smith.—In January, 1894, was sick. In March became jaundiced, and has remained so. About six weeks before admission his abdomen distended. On admission, November 6th, the liver could be felt most distinctly; it was uniformly enlarged, and extended almost to crest of ilium; gall-bladder felt as a smooth, round swelling. Jaundice; bile in urine; motions clay-coloured. December 6th, paracentesis abdominis to three pints. He began to be frequently sick, and died December 25th. Post-mortem: Liver very tough, hard and cirrhotic, bile-ducts much dilated; gall-bladder very distended. There was a hydatid cyst in the head of pancreas, three and a half inches by eight and a half inches; pancreatic tissue could be seen around it. It was pressing on and stretching the duodenum and bile-duct. It contained 250 c.c. of fluid, and before it was opened the common bile-duct was quite occluded, but after it was opened bile flowed freely.

HÆMORRHAGE INTO PANCREAS.

1894, 95. M., æt. 55. Dr. Newton Pitt.—Mitral stenosis. Tricuspid regurgitation. Nutmegged liver. There was a hæmorrhage, about one and a half inches across, in the tail of the pancreas.

1895, 433. M., æt. 17. Dr. Taylor.—A case of cerebellar tumour and hydrocephalus. There were many foci, and there was some hæmorrhage into substance of pancreas.

1897, 355. M., æt. 16. Dr. Washbourn.—Patient was admitted with all the symptoms of cardiac failure, from which he died. Twenty-two ounces of fluid were found in right chest, twelve in left. There were old pleuritic adhesions, with hæmorrhage into them. The lungs were such as might be found after long-standing heart disease, but all that was the matter with the heart was that it was dilated. There was a hæmorrhage in the middle of the pancreas.

LYMPHADENOMA OF PANCREAS.

1896, 55. M., æt. —. Dr. Taylor.—This was a case of Hodgkin's disease. The pancreas was much thickened and very hard. Its centre was occupied by a mass of hard, yellowish-looking growth, quite distinct from the pancreatic tissue. Histologically this was lymphoid.

CAVITY IN HEAD OF PANCREAS.

1891, 357. F., æt. 29. Dr. Hale White.—Admitted for profuse hæmatemesis and melæna which began eight days before admission. She was on admission, September 14th, very anæmic and had much pain after food, so the diagnosis was made of gastric ulcer. On 16th March, more hæmatemesis, and patient was so much blanched that laparotomy was done but it was found that adhesions prevented further operation. 21st: Again blanched and collapsed; three and a half pints of salt solution injected at 4.10 p.m., but died at 6.40. Temperature 105°. Post-mortem: Some peritonitis; stomach perfectly natural. Duodenum dilated and unusually adherent to pancreas; on its posterior wall, two inches from the pylorus, there was a small opening in which was a small clot of blood, the only extravasated blood found at the autopsy. This opening communicated with a smooth-walled cavity in the head of the pancreas into which neither vessel nor duct could be traced. There was no blood in the cavity although no doubt it was the source of the hæmatemesis. The pancreatic tissue around was indurated but the microscope did not reveal growth. There was no calculus. Dr. Shaw, who made the post-mortem, suggested the following sequence of events:—

1. Pancreatic calculus.
2. Dilatation of pancreatic duct.
3. Suppuration of around calculus.
4. Adhesion to duodenum with perforation.
5. Subsequent hæmorrhage from opening of a vessel in cavity.

ULCER OF DUODENUM ADHERENT OF PANCREAS.

1892, 243. M., æt. 61. Mr. Durham.—Hæmatemesis from a duodenal ulcer having the pancreas for its floor.

KIDNEY ADHERENT TO PANCREAS.

1896, 208. F., æt. 55. Mr. Golding-Bird.—Died after amputation of breast. There were a number of hydatids in the pelvis of the left kidney, which was consequently very enlarged, and the tail of the pancreas was firmly adherent to the left kidney.

ACTION OF ACID ON PANCREAS.

1896, 124. F. æt. 44. Dr. Washbourn.—She swallowed some hydrochloric acid at 10 a.m. on April 9th, and died four hours later. The autopsy took place twenty-four hours after death. No perforation of any viscus, but what little fluid there was in the peritoneum was strongly acid. The acid had a peculiar effect on the contents of the peritoneal cavity. The liver, spleen, and pancreas were hardened by its action, so that on section there were two distinct portions, the outer hard and leathery, the inner already decomposing and in the case of the liver and spleen, soft; in the case of the pancreas, stained by broken up blood.

ACCESSORY PANCREAS.

1896, 81. M., æt. 42. Dr. Hale White.—Died from aortic regurgitation. Close to the head of the pancreas lay a small elongated flattened mass, which on section had the structure of pancreas and was a small accessory gland.

SOME CASES OF EXOPHTHALMIC GOÏTRE ASSOCIATED WITH INCREASED INTRAOCULAR TENSION.

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AND

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INCREASED Intraocular Tension in association with Exophthalmic Goitre is a feature hitherto unobserved, or at least unrecorded, but at the same time one which has a most important bearing upon the "drug" treatment of this disease.

With a view therefore to directing attention to the occurrence, perhaps only occasional, of this condition, we have recorded in the following pages the salient points in the clinical histories of some cases that have recently come under our observation in the Ophthalmic Department, and have summarised the conclusions we have drawn from the study of these cases.

CASE I.

Helena S., æt. 24, domestic service.

First came under observation February, 1895.

1895	<p>Symptoms of asthenopia. Patient otherwise healthy.</p> <p>Vision and refraction:—</p> <p>R.E. = $\frac{1}{2}$ c $\frac{+ 0.5D \text{ sph.}}{+ 0.5D \text{ cyl. } 110^\circ} = \frac{1}{2}$; L.E. = $\frac{1}{2}$ c $\frac{+ 0.5D \text{ sph.}}{+ 0.5D \text{ cyl. } 130^\circ} = \frac{1}{2}$</p> <p>Tension normal. Fundus normal.</p> <p>Glasses prescribed, full correction for near work.</p>
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1896 January	<p>Complains of aching and pain in the eyes, especially after near work.</p> <p>Vision and refraction as in February, 1895, but with marked insufficiency of convergence for near work.</p> <p>Each eye, tension full, but falls after the instillation of Physostigmine. Field of vision complete to fingers.</p> <p>Ophthalmoscopic examination:— Deep central pitting of the discs. Marked venous pulsation.</p> <p>Treatment with weak Physostigmine drops suggested to the patient's medical man.</p>
February	<p>Each eye tension full or 1.</p> <p>Ordered Gtt. Physostigminæ Sulphatis (grs. ii. ad ʒi.) alternate nights.</p>
March	<p>Patient noticed that her eyes were becoming prominent, and that she had some difficulty in closing the lids. She also found that her throat was swelling slightly so that the collar of her bodice was uncomfortably tight.</p>
June 16	<p>Complains of severe pain in both eyes during the last week. Now the following signs of Graves' disease are noticed:— Pulse 102. Slight but distinct enlargement of both lobes of the thyroid. Symmetrical exophthalmos—retraction of the upper eyelids— Von Graefe's sign.</p> <p>Refraction as February, 1895. Vision (with correction), right eye = $\frac{6}{12}$; left eye = $\frac{6}{8}$ part; pupils active.</p> <p>Right eye tension 1, left eye tension 2. The tension falls under Physostigmine, but not to normal. No history of haloes.</p> <p>Field of vision contracted (<i>Vide</i> Chart I.¹).</p>

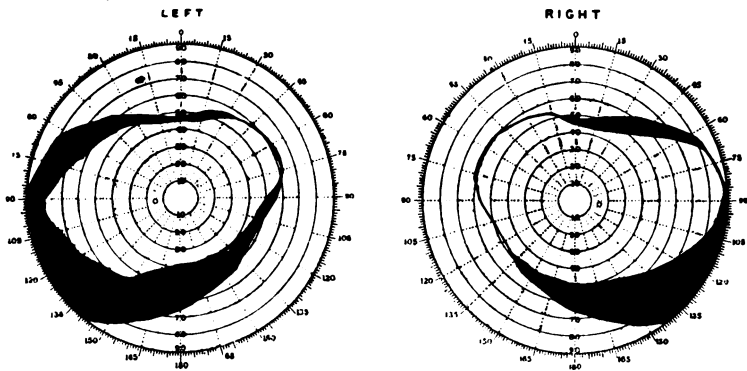


CHART I.

Ophthalmoscopic examination, as in January, 1896.

The Patient was admitted into Ruth ward, and kept under observation for a few days, when an upward iridectomy was performed on each eye. Patient made an uninterrupted

¹ In every chart the black area indicates the amount of loss of field of vision.

	recovery and was discharged July 3rd, 1896, the tension of each eye then being about normal, and the vision with correcting glasses = $\frac{6}{8}$ part.
July 7	Right eye tension normal. Left eye tension full.
Aug. 21	Right eye tension full. Left eye tension 1. Ordered Gtt. Physostigminæ grs. ii. ad $\frac{3}{4}$ j. o.n.
Aug. 28	Each eye tension normal.
Dec. 4	Each eye tension normal. Field of vision more contracted. (<i>Vide Chart II.</i>)

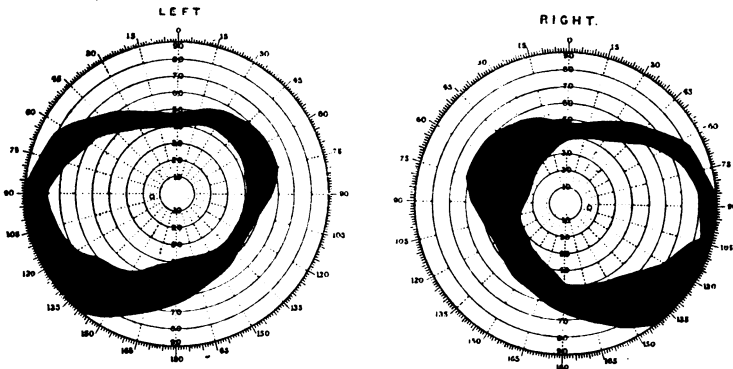


CHART II.

1897	During the year the patient attended the hospital eighteen times, usually at intervals of three weeks, and continued using the drops fairly regularly. If the drops were discontinued it was noticed that the tension rose, but it fell again to normal on resuming their use. The exophthalmos during this period became more marked, as did the hypertrophy of the thyroid.
1898 May 3	Patient did not attend between November 30th, 1897, and May 3rd, 1898. She then complained of great pain in the eyes. Each eye vision = $\frac{6}{36}$; with correcting lenses = $\frac{6}{18}$. Tension 1. Ophthalmoscopic appearances unchanged. Graves' disease appears stationary.
1899 February	Patient was now lost sight of for nearly a year. But in February, 1899, came to have her "drops" repeated. Refraction unaltered. Right eye vision = $\frac{6}{36}$ } with correcting lenses = $\frac{6}{18}$. Left eye vision = $\frac{6}{24}$

Each eye tension normal.

Field of vision much more contracted (*Vide* Chart III.).

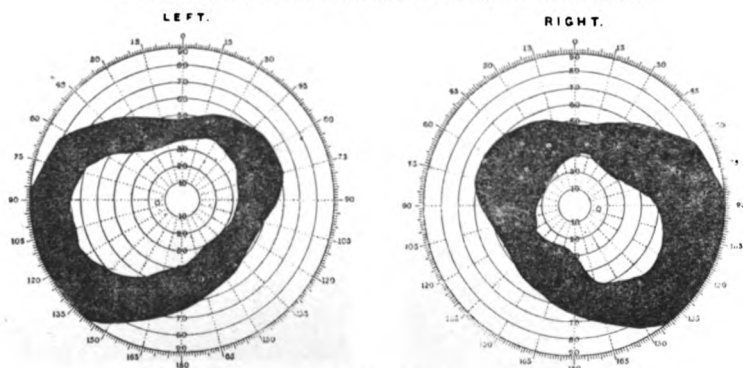


CHART III.

Ophthalmoscopic examination:—

Optic discs cupped about two-thirds of their extent, and overhung.

Marked venous pulsation.

Graves' disease is not so pronounced.

Pulse 120. Temperature normal.

Thyroid appears less enlarged, but on palpation a well-defined systolic thrill can be detected.

Neither the exophthalmos nor the retraction of the upper lids is so well-marked as formerly, but Von Graefe's sign is still present.

CASE II.

Elizabeth U., æt. 20, machinist.

First came under observation December 22nd, 1898.

1898
December

Complains of increasing prominence of the eyes, which she has noticed for the last seven months.

The following signs of exophthalmic goitre are present:—

Pulse soft, compressible, regular, varies between 106 and 158; temperature 99° F.

Hypertrophy of the lateral lobes of the thyroid, the right being more enlarged than the left.

Symmetrical exophthalmos—retraction of the upper eyelids—Von Graefe's sign present—Stelwag's sign present (*i.e.*, absence of nictitation).

Slight pigmentation of the skin of the lids.

Vision and refraction:—

Each eye vision = $\frac{5}{6}$ with + 0.5D Sph. = $\frac{5}{6}$.

Pupils active.

Tension 1.

No history of haloes.

Field of vision generally contracted (*Vide* Chart IV.).

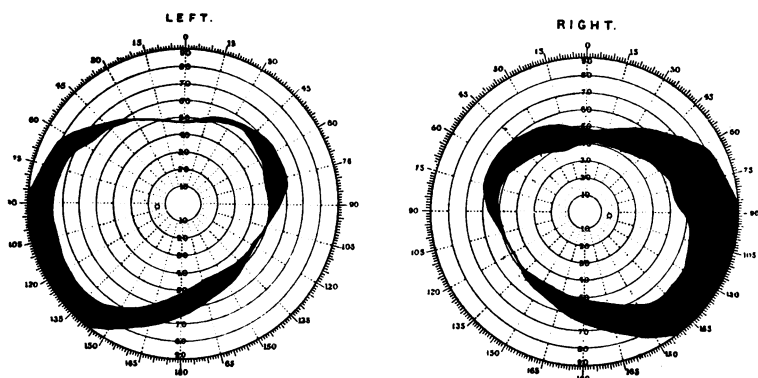


CHART IV.

Ophthalmoscopic examination :—

Deep central pitting of the discs, but edges not overhanging.
 Ordered Cocain., grs. ii., Physostig. grs. ii., Vaseline., ziv. , ft. ung.
 omnibus noctibus utendum.

1899	Each eye vision = $\frac{5}{8}$ part. Tension 1.
Jan. 6	Pulse 120. Temperature 99° F'.
Jan. 14	As January 6th.
Jan. 24	As January 14th. Ordered Vas. Physostigminæ as above, o.n.; and Gtt. Pilocarp Nit. grs. viii. ad $\text{z}.$, o.m.
Feb. 7	Field of vision still more contracted (<i>Vide</i> Chart V.).

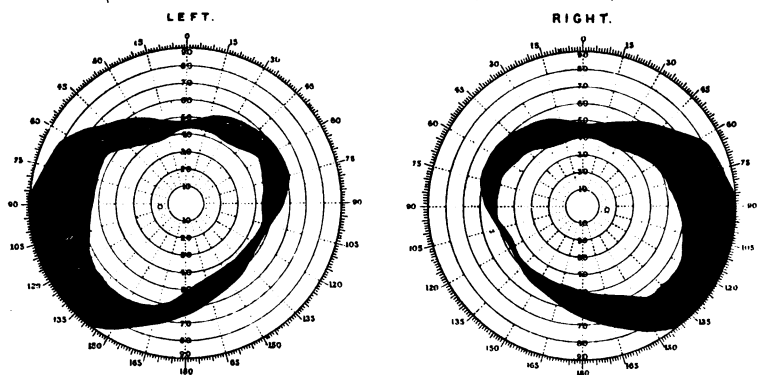


CHART V.

Feb. 14	} Each eye tension full to 1.
Feb. 21	
	As February 7th.

- March 3 Tension normal to full.
Pulse 106. Temperature 99° F.
Vas. Physostig. reduced to half strength. Drops to be continued.
- March 7 Pulse 132.
Tension: right eye full to 1; left eye normal to full.
Right field still more contracted; left, as on February 7th.
- March 17 Each eye vision = $\frac{5}{6}$ part, with + O. 5D sph. = $\frac{5}{6}$.
Tension 2.
Field of vision extremely contracted (*Vide Chart VI.*).

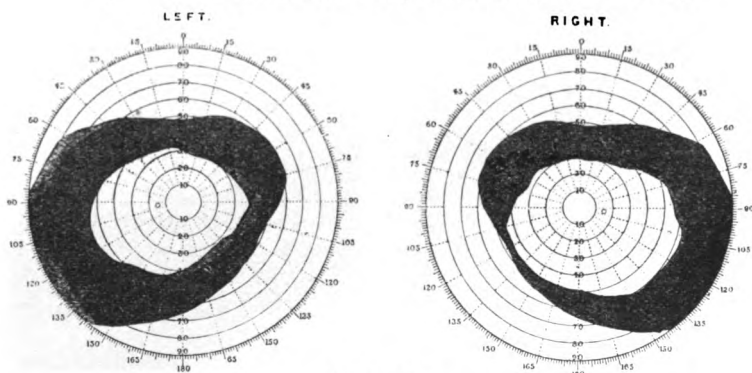


CHART VI.

Ophthalmoscopic examination:—

Optic discs deeply cupped (about one-third), edges overhanging.

Vessels pushed over to the nasal side, and pulsating.

On instilling Gtt. Physostig. grs. iv. ad $\frac{3}{4}$ i., the tension in each eye fell perceptibly, though not to normal.

Ordered Vas. Physostig. grs. iv. ad $\frac{3}{4}$ i., b.d.a.; and Gtt. Pilocarp. o.n.

- March 28 Each eye vision = $\frac{5}{6}$, with + O. 5D sph. = $\frac{5}{6}$.
Tension normal.
Field of vision practically normal.
- May 2 Each eye tension normal.

CASE III.

Maud P., æt. 18, dressmaker's apprentice.

First came under observation January, 1899.

- 1899
Jan. 20 Complains of "swelling of the eyes," first noticed a year ago; and failure of the sight of the left eye within the last few weeks.
The usual signs of Graves' disease are present, viz. :—
Pulse 120; temperature 99.4° F.; palpitations and dyspnoea after slight exertion.

Marked hypertrophy of both lateral lobes of the thyroid.

Exophthalmos (bilateral) — retraction of the upper eyelids — presence of Von Graefe's sign.

Pigmentation of the skin of the eyelids.

Vision and refraction :—

Right eye vision = $\frac{5}{6}$ (no Hm.); left eye vision = $\frac{1}{2}$ (no error of refraction).

Each eye tension full.

Field of vision complete to fingers.

Ophthalmoscopic examination :—

Marked pulsation of the vessels of the discs.

No cupping of the discs.

Ordered Vas. Physostig. grs. $\frac{1}{4}$ ad $\frac{3}{4}$.

Jan. 21 Patient was transferred to the Electrical Department under the care of Dr. Bryant. Here she received the continuous current for ten minutes each day, one pole being applied to the nape of the neck, the other over the thyroid. During this period the patient steadily improved. The exophthalmos became less marked, as did also the swelling of the neck.

Feb. 24 On February 24th, however, the patient presented herself in the Ophthalmic Department, complaining of pain in the eyes. On examination, the tension in each eye was found to be 1 at least; and the field of vision distinctly contracted (*Vide Chart VII.*).

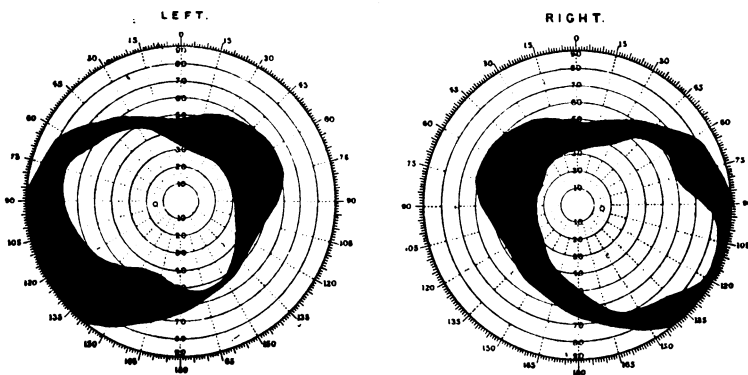


CHART VII.

Ophthalmoscopic examination :—

The pulsation of the vessels of the discs was more pronounced than before.

Ordered to use the Vas. Physostig. night and morning.

March 3 Each eye tension normal. Field of vision complete.

Right eye vision = $\frac{5}{6}$. Left eye vision = $\frac{5}{6}$.

- March 10 Vision as March 3rd.
 Right eye tension full. Field of vision slightly contracted on nasal side.
 Left eye tension normal. Field complete (*Vide* Chart VIII.).

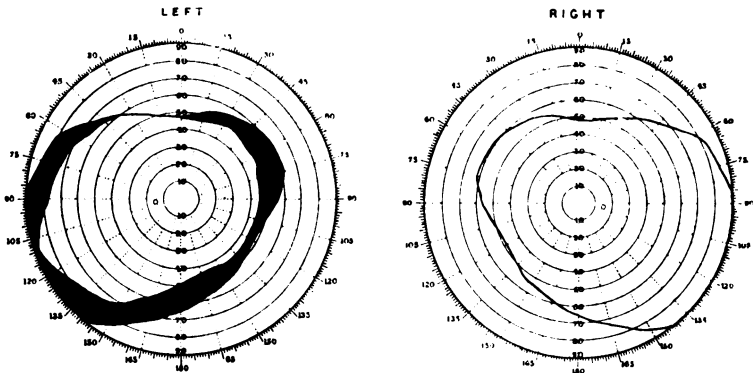


CHART VIII.

- March 17 Right eye vision = $\frac{5}{8}$, tension normal. * Field complete.
 Left eye vision = $\frac{5}{8}$ or $\frac{6}{8}$ (part), tension full to 1. Field slightly contracted (*Vide* Chart IX.).

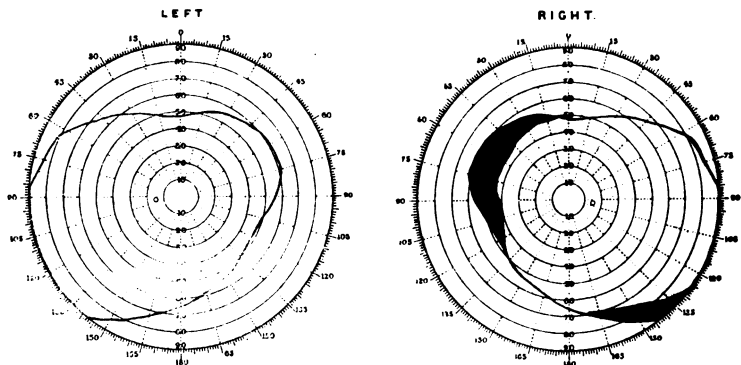


CHART IX.

Ophthalmoscopic examination :—

No cupping of the discs.

Marked pulsation of the vessels of the disc in the left eye,
 but none in those of the right.

Ordered Vas. Physostig. t.d.a.

- March 24 Each eye tension normal. Field of vision complete.
 April 28 Right eye vision = $\frac{6}{8}$, tension normal. Left eye vision = $\frac{6}{12}$, tension 1. Field of vision somewhat limited all round.

CASE IV.

Susan F., æt. 24, domestic service.

First came under observation January, 1899.

1899 Jan 31	<p>Complains of prominence of the eyes for the last two months; and pain in the left eye of a week's duration.</p> <p>Has worn glasses (right eye = - 3D cyl. axis 170°; and left eye = - 5D cyl. axis 0°) five years.</p> <p>Patient has the usual signs of exophthalmic goitre, viz.:—</p> <p>Palpitation and dyspnoea on exertion.</p> <p>Pulse, soft and compressible, varying between 82 and 118.</p> <p>Temperature 99·6° F. Subjective symptoms of heat, and flushings.</p> <p>Enlargement of the lateral lobes of the thyroid—the right being perhaps more marked than the left.</p> <p>Symmetrical exophthalmos—retraction of the upper eyelids—presence of Von Graefe's sign—absence of nictitation.</p> <p>Pigmentation of the eyelids and the surrounding skin.</p> <p>Vision, refraction, etc.:—</p> <p>Right eye = $\frac{6}{80}$ with $\frac{-0.5D \text{ sph.}}{-3D \text{ cyl. } 160^\circ} = \frac{6}{12}$; tension 1; pupil active.</p> <p>Left eye = $\frac{6}{18}$ with $\frac{-0.5D \text{ sph.}}{-1.5D \text{ cyl. } 0^\circ} = \frac{6}{12}$; tension 2; pupil active.</p> <p>Ophthalmoscopic examination:—</p> <p>Fulness and pulsation of the vessels of the discs.</p> <p>Discs otherwise normal, no cupping.</p> <p>Full correction ordered for constant use.</p>
Feb. 7	<p>Lids slightly full. Pulse 118. Temperature 99·8° F.</p> <p>Each eye tension 1. Field of vision slightly contracted.</p>
Feb. 14	<p>Right eye vision = $\frac{6}{24}$, tension 1. Left eye vision = $\frac{6}{36}$, tension 2.</p> <p>Ordered Vas. Physostig. gr. $\frac{1}{2}$ ad $\frac{3}{4}$ i. o.n.</p>
Feb. 20	<p>Field of vision still more contracted.</p> <p>Admitted into Ruth ward and treated by rest in bed, and the application of cold (Leiter's coil) to the eyes twice daily—for twenty minutes at each application.</p>
March 3	<p>Discharged from hospital. Condition much improved.</p> <p>Each eye tension normal to full. Field of vision when first taken is normal, but gradually contracts all round as the examination proceeds to almost half size.²</p> <p>Ordered to continue the use of Vas. Physostig.</p>
March 7	<p>Right eye vision = $\frac{6}{24}$ (with correction = $\frac{6}{12}$), tension full to 1.</p> <p>Left eye vision = $\frac{6}{18}$ (corrected = $\frac{6}{12}$), tension normal to full.</p>

² This spiral contraction appears to be due to exhaustion of the peripheral portions of the retina (and not to hysteria or simulation), and can only be demonstrated when the examination is prolonged over a considerable period.

March 14	Right eye tension normal. Field of vision as before. Left eye tension very slightly full. Ophthalmoscopic examination:— No venous pulsation.
March 21	Each eye tension normal.
March 28	Patient complains of seeing red and green rings round the light (with left eye only). Right eye tension normal, vision (with correction) = $\frac{6}{12}$. Left eye tension 2, vision (with correction) = $\frac{6}{18}$. Ophthalmoscopic examination:— Each eye marked venous pulsation. No cupping of the disc.
April 4	Patient now became inoculated with the Koch-Weeks' bacillus, and suffered from an attack of acute contagious conjunctivitis. This was treated with Sol. Protargol 1 per cent. ter. die.; and the use of the Vas. Physostigminæ was continued.
April 18	Right eye vision (with correction) = $\frac{6}{12}$, tension normal. Left eye vision (with correction) = $\frac{6}{12}$, tension 1.
May 19	Right eye tension normal. Left eye tension full.
May 23	Each eye tension normal.

CASE V.

Hilda D., æt. 20, domestic service.

First came under observation February, 1899.

1899 Feb. 7	Complains of headaches and pains in the eyes, after close work or reading; also that people accuse her of "staring." This last-mentioned symptom has only been noticed two months. The patient appeared to be in a very early stage of Graves' disease. Pulse 120. Temperature 99.2° F. History of palpitations on slight exertion. Hypertrophy of the lobes of the thyroid but very slightly marked. Slight exophthalmos, symmetrical—retraction of the upper eyelids—Von Graefe's sign present, but obtained with difficulty. Puffiness of the eyelids, with a peculiar reddish-brown pigmentation of the skin. Vision and refraction:— Each eye vision = $\frac{6}{8}$ (partly), no improvement with lenses. Tension normal or very slightly full. Homatropine (grs. iv. ad $\frac{3}{4}$ i.) instilled once, then— Right eye vision = $\frac{6}{8}$ (part) with + O. 5D cyl. vert. = $\frac{6}{8}$. Left eye vision = $\frac{6}{8}$ (part) with $\frac{+ O. 25D sph.}{+ O. 25D cyl. vert.}$ = $\frac{6}{8}$.
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Tension of each eye was now found to be very decidedly raised, nearly to 1.

Ophthalmoscopic examination:—

No cupping of the discs.

Pulsation of the vessels.

The full correcting lenses were ordered, and the patient told to return the following week. She did not, however, put in an appearance until six weeks later, when she came complaining of pains in both eyes.

March 21 On examination, pulse 144. Each eye tension 1. Visual acuity as before.

Fields of vision slightly contracted (*Vide* Chart X.).

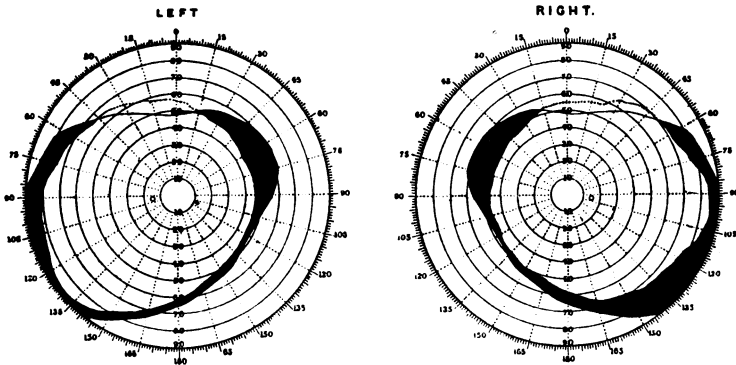


CHART X.

Ordered Vas. Physostig. grs. $\frac{1}{4}$ ad $\frac{3}{4}$ i. o.n.

CONCLUSIONS.

1. That a certain proportion of the cases of Graves' disease, presenting the usually recognised ocular symptoms, such as the lid signs, the proptosis and the swelling and pigmentation of the lids, are associated with increased intraocular tension.

2. That this tension is attended with the usual signs of high pressure, viz., pulsation of the retinal veins, cupping of the optic disc, limitation of the field and diminished acuteness of vision.

3. The high tension is usually bilateral, but it often varies in a very marked manner, so that it may be normal for a time in one or other eye.

4. Its tendency is to fall ultimately to normal, but it may last for months or even for years.

5. The sight generally returns after the removal of the pressure, the recovery being due to the elasticity of the tunics of the eye in the usually young subjects of this disease (with us nearly all young girls).

6. This same elasticity makes the cupping of the disc quite a minor feature. There is often deep central pitting, but it generally implicates less than one-fourth of the disc.

7. The rise of tension is probably merely a result of the nervous and vascular disturbances that are at the root of the malady.

8. The tension is difficult to appreciate and to estimate, on account of the soft thickening of the lid tissue covering the globe, and of the tissues of the orbital floor supporting it, and on account of the softness of the ocular tunics. It is appreciated more certainly if palpation is made from the sides, so that one finger estimates the hardness of the globe when the other supports it against the pressure.

SUPPURATIVE PYLEPHLEBITIS.

By J. H. BRYANT, M.D.

I HAVE performed necropsies on two cases of this obscure disease during the last six months. Both presented many curious features and points of interest, and neither was correctly diagnosed before death. I looked up our records for similar cases, and was so much impressed by the comparative rarity of its occurrence, viz., eleven cases during the last twenty years, that it must serve as my apology for writing this paper.

This paper is based on twenty cases, nine of which were reported by Dr. Carrington, in the year 1883, in the Guy's Hospital Reports, in a paper entitled Multiple Abscess in the Liver, and eleven cases which have been examined in the post-mortem room during the last twenty years. I am much indebted to Mr. J. A. Butler for his help in looking up the reports, and to my colleagues for allowing me to use their cases.

Ætiology.—Suppurative pylephlebitis is nearly always caused by a lesion in one of the organs drained by the portal system, *e.g.* :—

1. Appendicitis in eight out of the twenty cases, or 40 per cent.
2. Gastric ulcer, 1.
3. Duodenal ulcer, 1.
4. Ulcer of the rectum, 1.
5. Dysenteric ulceration of the colon, 1.
6. Gall-stones and suppurating gall-bladder, 2.
7. Pyosalpinx and suppurating ovary, 1.
8. Abortion, 1.
9. Cause doubtful in 2.

¹ Davidson mentions—

10. Sloughing of the cæcum.
11. Abscess of the spleen.
12. Ulceration of common bile-duct extending to the portal vein.
13. Ulceration originating in the mesenteric vein involving the vena portæ.
14. Dysentery (rare).
15. Inflammation of the umbilical vein in infants.

² Pepper mentions—

16. Thrombosis and suppuration following operations for hæmorrhoids.

³ Fagge mentions—

17. Suppurating mesenteric glands.

⁴ Frerich reports a case of—

18. Fish-bone perforating the mesenteric vein.

⁵ Wilks mentions—

19. Submucous abscesses of the rectum implicating the hæmorrhoidal veins.

Sex appears to have very little influence—eleven of the cases were males and nine females.

Age.—The ages varied from fifteen to sixty-two.

8 occurred in the 2nd decade.

5	"	"	3rd	"
2	"	"	4th	"
4	"	"	5th	"
0	"	"	6th	"
1	"	"	7th	"

The average age worked out at twenty-nine.

It is interesting to note that the onset in two of the cases (2 and 6) was ushered in by a chill.

Morbid anatomy.—The body was noted as being wasted in eleven, *i.e.* in 55 per cent. of the cases.

The Portal vein.—The main vessel may contain thick, sanious pus and broken up blood-clot. The clot is very rarely adherent to the intima, and is usually of a greyish or pinkish-yellow colour. In some cases the main vessel does not contain clot or pus, but

¹ *System of Medicine.* Allbutt, vol. iv., p. 128.

² *System of Medicine.* Pepper, vol. ii., p. 1098.

³ and ⁴ *Fagge's Principles and Practice of Medicine.* Vol. ii., p. 498.

⁵ *Pathological Anatomy.* Wilks and Moxon, p. 462.

both pus and septic thrombi may be found in its branches in the liver; in other cases septic thrombi may be found in all three, viz., the portal vein, its radicles and its branches.

In case 1.—Pus and septic thrombi were found in the main vessel and its branches.

2.—The portal vein was filled with a soft septic thrombus. The splenic vein was full of thick greenish-coloured pus, and the small veins forming it were nearly all filled with a soft septic thrombus.

3.—A recent thrombus partially obliterated the lumen of the lower branch to the left lobe.

4.—The terminal branches were thrombosed.

5.—Partially decolorised, softening clot in the main trunk of the portal vein.

6.—Septic thrombosis of the superior mesenteric vein extending into the portal vein and its main branches.

7.—Portal canals full of foul pus.

8.—Pus in the smaller branches of the portal vein only.

9.—A suppurating thrombus in the superior mesenteric vein which could be traced into a mass of purulent material around it. The branches of the portal vein were filled with pus, and the main trunk was obliterated by a firm thrombus.

10.—Two drachms of pus were found just beyond the bifurcation of the portal vein, and the intima of the vein was shaggy from adherent lymph.

11.—Portal vein completely blocked by a soft clot which contained yellow pus in its centre. The splenic vein was plugged and full of pus.

12. (Carrington 1.)—"Pus in the portal vein and its branches. The portal veins were extremely diseased throughout, being in a state of intense suppurative inflammation. They were much dilated, quite distended with a pure pus of a yellowish colour. Nearly the whole of the liver was thus affected. A section in any part which cut through the portal vessels showed a purulent fluid running from the vein. Not merely did they contain pus but their coats had been subject to a severe inflammation. Thus the walls were softened and thick, and in parts detached from the

hepatic tissue around. In the large trunks there were distinct patches of lymph adherent to the coats, and in the trunk itself of the vena portæ there was a firm layer of pink-coloured lymph closely adherent to the wall.

13. (Carrington 2.)—In some parts of the right lobe the suppuration had advanced as far as the capillaries and the secretory system, so that a section of the organ displayed the lobules distinctly mapped out, the lobules themselves being of a yellowish colour from the suppuration going on within them, and the hepatic vein still visible as a red spot in the middle. In some few places the suppurating lobules had run together, and having become much softened would in a very short time have formed distinct abscesses. A most remarkable fact was that one of the larger trunks of the portal vein was quite unaffected, yet when traced upwards its branches were found distended with pus. It appeared from this that the inflammatory process had been primarily and mainly in Glisson's capsule, and that the tubes had been afterwards involved, and perhaps from the periphery towards the trunks.

14. (Carrington 3.)—"The main branches of the right lobe were partially plugged by breaking down blood-clot. This state extended to the second and third sub-divisions; then stopped; and the remainder of the vein was healthy for some distance till the more terminal part was approached."

15. (Carrington 4.)—Pus in the branches of the portal vein.

16. (Carrington 5.)—Diffuse phlebitis of the mesenteric veins, which were full of pus. Portal vein plugged with a firm cylindrical clot, smoothly channelled in its centre, which had been probably formed by lamination on the vein wall. The main branches were full of grumous pus and blood-clot, and the smaller branches contained pus.

17. (Carrington 6.)—Large branches of the portal vein filled with pus.

18. (Carrington 7.)—Suppuration along a small portal canal.

19. (Carrington 8.)—Portal vein as thick as one's little finger and distended with thick puriform thrombus. The branches of the vein were also full of pus.

20. (Carrington 9.)—The main trunks were not affected.

The liver is usually slightly and uniformly enlarged. The greatest weight recorded was ninety-eight ounces, and the lowest forty-two ounces, the average worked out at sixty-six ounces. The enlargement was sufficient in 60 per cent. of the cases to allow of the liver being detected below the costal margin by palpation. In ten of the cases the liver is noted as being adherent to the diaphragm; no mention is made of adhesions in eight, and there were no adhesions in two. It is quite the usual thing for the inflammatory process to spread to the surface of the organ and to set up a local or even general perihepatitis, followed by fibrous adhesions, fixing the organ to the under surface of the diaphragm. Case 1 was remarkable in this way, the liver was double its normal weight, was much larger than normal, was firmly adherent to the diaphragm, and yet did not extend beneath the costal margin. The probable explanation is, that the lower part of the organ, before it commenced to enlarge, became adherent to the lower and inner border of the thorax, and so when it commenced to enlarge, the adhesions prevented it from increasing in size in a downward direction. The surface of the organ is usually smooth except for the patches of perihepatitis. Rarely the abscesses project and form small rounded yellow nodules covered by recent lymph. In all the cases abscesses were found in the liver; these abscesses vary somewhat in size and distribution; the whole organ may be studded with points of suppuration much smaller than a pea; the points of suppuration may run together and form larger abscesses, which may get as large as a walnut or even larger; one lobe may be much more affected than the others, and finally, the abscesses may be confined to the area supplied by quite a small portal vessel. Dr. Goodhart has recorded one case (No. 10) in which a good deal of healing had taken place.

It is common for the inflammatory process to spread through the diaphragm and to set up pleurisy at the right base; it was noted in eleven of the cases; effusion was present in five of the eleven cases, and the effusion was purulent in three.

The spleen may or may not be enlarged; it was stated to have been enlarged in six of the cases; the highest weight recorded

was twenty-three ounces, and the lowest four ounces; it was described as normal, soft, and pulpy; abscesses in its substance were noted in two of the cases (7 and 11), and it was lardaceous in one (11). Old or recent capsulitis is common. Infarcts were found in one of the cases (20, *i.e.*, Carrington 9).

The pancreas was generally found to be healthy; multiple abscesses were found in one case (5), and an abscess posterior to it in one case (12, *i.e.*, Carrington 1).

Peritonitis was common, *viz.*, in 50 per cent. of the cases, and could generally be traced to the primary condition, *e.g.*, four to appendicitis, one to pyosalpinx, one to abortion, and one to post pancreatic abscess.

Pathology.—The condition is due to an invasion of the portal vein by pyogenetic micro-organisms. This may be caused by some ulcerative or suppurative lesion in one of the organs drained by the portal system, and may be brought about in a variety of ways, or may be caused by direct implication of the portal vein or one of its branches.

1. A small vein may be injured from adjacent ulceration or suppuration, thrombosis follows, the thrombus becomes infected with pyogenetic micro-organisms, it then spreads to a larger vein and then to the portal vein and its branches in the liver; at this stage the portal vein, its branches, and certain of its radicles would be full of pus and soft breaking-down thrombus.

2. The thrombus in the vein primarily involved may break down, and the fragments containing pyogenetic micro-organisms may be carried to the smaller branches of the portal vein and the portal capillaries in the liver, colonies of the micro-organisms develop, give rise to inflammation, and the formation of septic thrombi which may spread in both directions and may be followed by necrosis of the hepatic cells and suppuration.

3. Masses of pyogenetic micro-organisms may be carried from the primary lesion and give rise to capillary emboli, followed by thrombosis, which may spread backwards, until fairly large branches of the portal vein are involved. This offers an explanation for those cases in which only the branches of the portal vein in a certain part of the liver are affected.

4. The portal vein or one of its branches may be directly involved, *e.g.*, in cases of gastric ulcer and suppurating gall-bladder. In the former case the ulcer may become adherent to the liver, cause the formation of a local abscess, which may open up a branch of the portal vein, and so infect it and lead to a spread of the infection along its branches in the liver and back along its main trunk to the portal vein itself. A suppurating gall-bladder may act in much the same way. I remember performing a necropsy on such a case at the St. George's Union Infirmary. The gall-bladder contained gall-stones and was suppurating, the anterior wall attached to the liver had given way, an abscess about three-quarters of an inch in diameter had formed in the adjacent liver substance, and the portal canals in the immediate neighbourhood were full of pus on account of the abscess directly invading a portal canal. There were multiple abscesses throughout the liver, especially in the right lobe in the neighbourhood of the gall-bladder.

5. When disease of the pelvic organs is the primary cause, pelvic peritonitis is usually present, and this may lead to septic thrombosis of the inferior hæmorrhoidal veins or other branches of the inferior mesenteric vein.

6. Occasionally no cause can be found. In case 2 there was no lesion of any organ drained by the portal circulation, the primary disease was an empyema, which had opened into the lung, and at the time of the autopsy I suggested two possibilities to explain the condition.

- (1) That a considerable amount of the pus coughed up from the empyema may have been swallowed, and the pyogenic micro-organisms have entered the portal system by absorption from the stomach and intestine.
- (2) That the exploratory punctures which perforated the diaphragm, may have punctured the liver and inoculated some of the portal canals directly from the empyema.

In case 12 (Carrington 1), the first case described in Dr. Carrington's paper, the abscess behind the pancreas might well

have been the primary cause of the suppurative pylephlebitis by first of all causing phlebitis and then thrombosis.

One of the most extraordinary facts in connection with this disease is its absence as a complication of typhoid fever, which is by far the most common form of ulceration of the intestine met with in this country. I have not been able to find one single case in our records.

Bacteriology.—The bacillus coli communis in pure culture was found in case 2; streptococci and a bacillus not identified in case 1; a diplococcus (not identified) from case 3. Staphylococci have been found, and in cases complicating dysentery the amœba coli.

Histology.—Multiple abscesses in the liver. The portal capillaries and branches of the portal vein are filled with pus. In specimens stained for micro-organisms large colonies of bacilli are found in the capillaries and veins, in some places spreading into the hepatic tissues, where necrosis of the hepatic cells and small-celled infiltration can be seen.

Symptoms and physical signs.—The onset, as far as can be judged from a careful analysis of the histories of the cases, is often acute, for 60 per cent. of the cases began in this manner.

A striking example is case 1; the boy is stated to have been at work in his usual health on the day before the illness, which commenced suddenly at 1 a.m. with vomiting and pain in the right side. The initial symptom in five of the cases was vomiting; in three, a rigor; in two, pain in the right side; in two, abdominal pain; in one, pain in the back; in one, pain in the loins; in one, diarrhœa; and in one, acute pain in the loins. It appears from the above analysis that many of the cases have a very definite onset.

The prominent symptoms for which admission was sought were: two for pain in the right side of the chest and pyrexia, three for abdominal pain, two for pain in the right hypochondriac region and jaundice, for vomiting and pyrexia, for biliary colic, for diarrhœa and abdominal pain, for jaundice, for pyrexia, and for discharging sinus in the groin. In six of the cases no mention was made.

Vomiting is one of the most marked and constant symptoms, it was mentioned as occurring in 60 per cent. of the cases. The vomit does not show any particular characteristic.

Diarrhœa is another common symptom, it occurred in 45 per cent. of the cases.

Constipation, however, is sometimes present, it was noted in 35 per cent. of the cases.

Pain is a very important symptom and is more constant than any other with the exception of pyrexia. Pain was noted in all the cases with the exception of two, in which no mention of its presence or absence was recorded. It is usually situated in the right hypochondriac region; it may also be in the epigastrium, in the right side of the chest, in the umbilical region, and may even be felt across the lower part of the abdomen.

Sweating was noticed in only 35 per cent. of the cases, and is not such a characteristic symptom as the text-books would lead one to suppose.

Rigors were noted in 50 per cent. of the cases, and they were definitely stated not to have occurred in 40 per cent. of the cases.

Jaundice is also a variable symptom, it occurred in 40 per cent. of the cases, and in 55 per cent. a definite note is made of the absence of this symptom. Pepper states that it occurs in 75 per cent. of the cases, and Fagge writes that it accompanies pylephlebitis oftener than single abscess.

Delirium may occur and is mentioned as being present in three of the cases. It is not usually an early symptom.

Collapse was noted in four of the cases.

The temperature.—The highest temperature recorded was 107° and the lowest 96°. It does not run any particular course and may be continuous in character—*vide* chart, case 1, which is almost identical with the typical pneumonia chart depicted in Taylor's Medicine; it may be somewhat typhoidal in character—*vide* chart, case 2. It may be intermittent or remittent—*vide* charts 8 and 9.

The pulse is generally rapid, from 100 to 140, well above the usual physiological ratio to the temperature, and is soft and compressible.

The *respiration* rate is also increased, and it is high, chiefly on account of the frequency with which the right pleura is affected.

The aspect of the patient.—The patient lies on his back and is weak, wasted and anæmic, looks very ill, has sunken eyes and a pinched and anxious expression, is sometimes jaundiced, may be restless and complain of pain over the hepatic area, and the cheeks may be flushed. Osler describes the complexion as being muddy. The *skin* may be moist or hot and dry. It was, for example, always hot and dry in case 1; it may be jaundiced.

There are often sordes on the lips and teeth.

The abdomen may or may not be distended. Distension when present may be associated with the condition which causes the disease. A slight fulness or even a marked swelling may be noticed in the right hypochondriac and epigastric regions. On palpating the abdomen an increased sense of resistance, or even marked rigidity may be appreciated in the right hypochondriac and epigastric regions.

The liver can usually be felt to be enlarged and its edge may be palpated just below the costal margin, or even as low as the level of the umbilicus. The edge is often sharp and well-defined. The surface is nearly always smooth and regular, occasionally, however, small soft and even fluctuating nodules may be felt, when the abscesses are large enough to project from the surface of the organ. The liver could be palpated in 60 per cent. of the cases.

Pain and tenderness in the right hypochondriac region is present in the majority of the cases.

The *Spleen* may or may not be felt. If enlarged, it is in most instances due to the toxic condition of the blood and not to a passive congestion, as the portal vein is hardly ever, if ever, completely obstructed by a thrombus in this disease. When an abscess in the spleen is the primary cause of the disease, the organ is tender, a fluctuating swelling may be felt, and on auscultation a rub may be heard over it.

If there is a history of an attack or attacks of appendicitis, on palpation, an indefinite tumour or sense of resistance may be felt in the right iliac region, due to thickening and matting of the parts.

The Thorax.—The lower part of the thorax on the right side may show distinct bulging. It was described in three of the cases. In case 1 it was extremely well marked and noticeable: the intercostal spaces were also filled out when compared with the spaces on the left side. In the above mentioned case it was a most misleading sign on account of the liver not being felt below the costal margin. The bulging was general, and it did not suggest any local enlargement of the liver.

In one of the cases a "falling in" of the affected side of the thorax was noted.

Pleurisy affecting the right lower lobe is common. It may be a simple dry pleurisy causing pain, impairment of movement and a pleuritic rub at the right base, or, there may be serous or purulent effusion, causing impairment of movement, displacement of the cardiac impulse, diminished tactile vocal fremitus, dullness, deficient entry of air, distant bronchial breathing at the level of the fluid, and diminished voice-sounds or ægophony.

The cardiac impulse, dullness, and sounds are usually normal.

The urine may be normal. The specific gravity may vary from 1010-1030, the reaction is acid, albuminuria may be present, it was noted in four of the cases, the chlorides were stated to have been diminished in one of the cases, and bile pigment was noted in the cases in which jaundice was a symptom.

The fæces were described as steel-grey-coloured and well formed (8); well formed and typhoid in character (9); light clay-coloured (12, Carrington 1); dirty-white (19, Carrington 8). Dr. Goodhart made an interesting statement in connection with case 8, viz., "That bile in the motions without diminished jaundice pointed to a collection of small abscesses in the liver."

The duration is very variable. As far as could be judged from an estimate of the symptoms and history, the shortest was 5½ days and the longest 296. The average works out at 56 days. Excluding the exceptional case which lasted 296 days the longest was 88 days, and if the average is taken of these 19 cases it is 43 days.

Case 2, the duration of which was 296 days, is extraordinary on account of the morbid changes found in the liver, there being

very marked signs of a healing and cicatrizing process having occurred. I have not been able to find any similar case recorded.

The prognosis is extremely bad, a correct diagnosis is, practically speaking, a death warrant. I have not been able to discover a single authentic case of recovery from this disease.

The diagnosis is difficult. A correct one is rarely made. Out of the twenty cases only two were accurately diagnosed before death. Diagnoses of gastro-enteritis, pneumonia, empyema, septic pneumonia, gall-stones, biliary colic, enterica, appendicitis and hepatic abscess, spinal caries, rupture of a reduced hernia, and subdiaphragmatic abscess were made.

Pyrexia, rigors, sweating, rapid pulse, abdominal pain, and tenderness in the right hypochondriac or the epigastric region together with uniform enlargement of the liver and tenderness of that organ, especially if associated with or following any ulcerative lesion of the alimentary tract below the œsophagus, should suggest the possibility of suppurative pylephlebitis. If *pleurisy* or *pleurisy with effusion* happens to be a complication it may be considered to be the primary disease, and suppurative pylephlebitis may be overlooked. If the thorax is bulged, and the liver tender and enlarged; if there is a history of rigors and sweating; if there is jaundice, vomiting and diarrhœa; then suppurative pylephlebitis must be seriously taken into consideration.

Case 1 was diagnosed pneumonia: the acute onset, the short duration of the disease, the absence of sweating, the hot dry skin, the continuous temperature which was almost identical in character with the typical temperature chart of lobar pneumonia depicted in Dr. Taylor's book on Medicine, the sudden drop to 96° on the eighth day which simulated a crisis, the dulness and bronchial breathing at the right base, all pointed to pneumonia as the most likely diagnosis. Against pneumonia, however, was the absence of cough and expectoration, the wasting, the sunken eyes, the general aspect of the patient, and the bulging of the lower part of the thorax on the right side. The fact also that the liver could not be felt was much against a primary disease of the liver or subdiaphragmatic suppuration.

The fact that vomiting and diarrhœa were so marked during the first few days of the illness in case 1 was the origin of the diagnosis of gastro-enteritis.

Enterica must be distinguished by the pulse, temperature, and respiration ratio which is so characteristic of this disease, *e.g.*—

		Temperature.	Pulse.	Respiration.
Physiological ratio	...	104°	125	33
Typhoid fever	...	104°	80	28
Pneumonia	...	104°	100	40-50
Pylephlebitis (case 1)...		104°	140	44

From the above comparison the extraordinary low ratio of the pulse in typhoid fever is brought out—this ratio is more or less well marked in the greater number of cases of enterica.

The spleen may be enlarged and the abdomen distended in both diseases. Jaundice is very rare in typhoid fever, and sweating is another symptom which rarely occurs in this disease. The presence of the characteristic rash would point to typhoid fever. The examination of the blood is of the greatest importance: a positive Widal reaction would point to typhoid fever or a previous attack of this disease.

The *treatment* unfortunately is only symptomatic. Pain may be relieved by local applications, such as fomentations, poultices, &c., and by the internal administration of opium and by subcutaneous injections of morphia. The general condition of the patient should, if possible, be improved by a nourishing diet, by stimulants, and by drugs, such as quinine, strychnine, iron, &c. The only possible hope of permanent cure in the present condition of our knowledge would be an early diagnosis not only of pylephlebitis but also of its cause, and an immediate eradication of that cause by operation, should it be practicable.

LIST OF CASES.

CASE 1.—SUPPURATIVE PYLEPHLEBITIS.—PYÆMIC ABSCESSSES IN THE LIVER
OLD APPENDICITIS. PLEURISY WITH EFFUSION.

Clinical, No. 444. 1899. (Refers to the number of the Report.)

A. E. F., æt. 15, admitted under the care of Dr. Bryant into Clinical ward on August 30th, 1899, for vomiting and pyrexia. On August the 22nd he went to work and seemed in his usual health. At 1 a.m. on the 23rd he vomited and complained of pain in the right side. He felt feverish, but had no rigors. He had eaten some green apples before going to bed. He did not remain in bed, but as he did not improve he was taken to see a doctor, who diagnosed gastro-enteritis and ordered him to be kept in bed on a milk diet. He was very sick again on the 28th, and as the sickness continued, and he seemed worse, he was admitted on August 30th. The bowels throughout have been freely opened and the motions were loose. There has been no cough and no expectoration.

Several years ago he was treated in King's College Hospital for a tuberculous ankle, and was kept in bed for some months. He was also treated at the same hospital for some disease of the left eye, for which iridectomy was performed. Two years ago he was treated for "appendicitis" by the same doctor, who sent him up for admission.

Condition on admission.—Temperature 101·6°, respiration 48, pulse 120. A fairly well-developed boy. He seemed rather collapsed. His face was flushed, his eyes sunken, his lips cyanosed, and his skin hot and dry.

Respiratory System.—Respirations shallow, regular, 48. The lower part of the chest on the right side was bulged, the intercostal spaces were filled out, and this side of the chest did not move well on respiration. The T. V. F. was increased over the right lower lobe behind. There was absolute dulness on the right side, from the fourth space in front, and it extended round the chest in practically a straight line, being a little lower behind than in front. On auscultation the vesicular murmur was found to be absent over the dull area, and over the lower part behind, *i.e.*, below the ninth rib; along this there was well-marked bronchial breathing (*vide* Diagram at the end of this Paper), and the voice-sounds were rather ægophonic in character. Above the upper limit of dulness a few medium-sized non-consonating râles were audible. There was no cough and no expectoration.

Circulatory system.—Pulse 120, low tension, and running in character. The cardiac impulse was in the fifth space in the nipple line. Cardiac dulness normal. Sounds normal.

Alimentary system.—Tongue dry and coated with brown fur. Sordes on the lips and teeth. The abdomen moved well on respiration. It was a little rigid in the upper part, and there was slight tenderness on palpation below the right costal margin. The liver and spleen could not be felt. There was no abnormal area of dulness. There was no tenderness and no tumour to be felt in the right iliac region.

Urine 1023, dark coloured, acid. No albumen, chlorides not diminished.

He was thought to be suffering from lobar pneumonia of the right lower lobe, complicated by some obscure hepatic or subdiaphragmatic condition, to account for the curious conformation of the right side of the thorax.

Progress.—At 9 p.m. on the day of admission his temperature reached 104.6°, and he was sponged.

August 31st. At 10.30 p.m. he commenced to vomit, and the vomiting continued until 4 a.m.

September 1st. He was given minim doses of Tinct. Iodi every half hour and the vomiting ceased. At 7 a.m. he collapsed, and became cyanosed and almost pulseless. At 10 a.m. his temperature was 96°. On account of the bulging of the thorax on the right side, and the filling out of the intercostal spaces, the question of empyema, subdiaphragmatic abscess, or hepatic abscess all along had been considered, and it was deemed advisable to explore. No pus was found. Oxygen was administered, also musk and brandy, after which his condition improved for a time. About 12 p.m. vomiting came on again but was checked by Tinct. Iodi. The temperature rose to 103.6° and patient sank and died at 10 a.m.

Necropsy (performed by Dr. Bryant), No. 308. 1899. Body wasted. In the right pleural cavity about a pint of clear serous fluid was found. The base of the right lung and the upper surface of the diaphragm were covered with recent lymph. The right lower lobe was quite airless from compression. There was no pneumonia. The heart weighed six ounces. It was healthy.

The liver was firmly adherent to the diaphragm and to the parts which were in relation to it. The adhesions were firm and fibrous. It was much enlarged and its upper level reached as high as the third rib. The lower edge did not project below the costal margin. It weighed seventy-two ounces, that is, just double what it should have weighed. *In situ* it looked as if it had been dragged upwards by means of its adhesions to the diaphragm, and possibly by actual permanent contraction of the diaphragm. It felt softer than normal. The capsule was a good deal thickened. On section, multiple small foci of suppuration were seen, more particularly in the upper part of the right lobe, where they had run together in places. Throughout the right lobe little abscesses and collections of abscesses were seen. The left lobe was comparatively free. The portal vein and its branches contained sanious pus and ante-mortem thrombus. It was nowhere completely obstructed.

The spleen weighed four ounces and was firmly adherent to the under surface of the diaphragm. The cause of the portal pyæmia was found in the appendix. The cæcum and appendix were bound down by firm, thick, connective tissue. On making a careful dissection the appendix was found curled up in the form of the letter O, and lying in the centre of the coil was an oval concretion, which had ulcerated through the appendix, the hole in which was found. Another opening was found in the centre of the coil communicating with the cæcum, and, I take it, through the latter opening the pus of the appendicitic abscess had discharged itself into the cæcum. The other abdominal viscera were healthy.

Bacteriology.—A specimen of pus was obtained from the liver for bacteriological examination, every precaution being taken to prevent the possibility of accidental contamination. Microscopical examination showed the presence

of a few short bacilli. Cultures showed the presence of the bacillus coli communis. No other organism was found (*vide* also Chart I. at end of Paper).

CASE 2.—SUPPURATIVE PYLEPHLEBITIS.—MULTIPLE ABSCESSES IN THE LIVER FOLLOWING EMPYEMA.

DR. HALE WHITE, No. 11. 1899.

J. R., æt. 44, a publican, was admitted under the care of Dr. Hale White on November 9th, 1898, for pain in the right side and pyrexia. About a month before admission he had been swimming in the sea at Margate when he suddenly lost consciousness and sank. He was rescued, but did not recover consciousness for about half an hour. A week later he suffered from slight shortness of breath, and a cough which caused pain on the right side of his chest. His sputum was occasionally of an unpleasant taste. His breathing, cough, and the pain gradually became worse, and on November the 7th he called in a doctor. On the 7th and 8th he coughed up nearly a pint of yellow sputum tinged with blood, and the right side of his chest became extremely painful. Since November 6th his voice had been hoarse. He was advised to come into the hospital for treatment.

The family history was good. He had been rather a heavy drinker, and had occasionally suffered from quinsy, but otherwise had always had good health. He was married and had two healthy children.

Condition on admission.—Pulse 100, respiration 32, temperature 103°. He appeared healthy and well nourished; his breath smelt foul, he was very hoarse, and he occasionally coughed up yellowish sputum of a very foul odour. He was able to lie upon his back or right side, but not upon his left side.

Respiratory system.—The left side of his chest moved better than the right, and there was some falling-in noticed on the right side from the sixth rib to the costal margin. The T. V. F. was diminished on the right side both in front and behind, and in front was completely absent below the fifth rib. Slight impairment of resonance was noticed on the right side above the fourth rib, and below it an area of complete dullness which extended behind below the angle of the scapula a little beyond the scapular-line, but did not reach the spines. It was difficult to make out the limits of the dull area accurately because of the pain which palpation and percussion caused. The breath-sounds were harsh on the left side, and on the right side both voice and breath sounds were absent over the dull area, while above the fourth rib in front there were small consonating râles, and along the right axillary border "sticky" râles were heard with inspiration.

Circulatory system.—The cardiac dullness and sounds were normal, and the impulse was felt in the fifth space half an inch internal to the nipple-line.

Abdomen.—The liver could be felt about one inch below the costal margin, and palpation caused pain. The spleen could not be felt.

Urine.—Sp. gr. 1030, acid, no albumen, no sugar, no blood. Carbolic acid was present.

Progress and treatment.—The treatment ordered was Capsulæ Creosoti mii. t.d.s. M. Scillæ Ammon. ʒj. t.d.s.* Steam kettle with Tinct. Benzoini Co. ʒj. and Pil. Col. et Hyosc. gr. viii. h.n.

The free expectoration of the very foul-smelling yellowish sputum was slightly diminished and the tenderness was less. On November 12th Dr. Hale White

* For the composition of the Mixtures, etc., mentioned, *vide* The Guy's Hospital Pharmacopœia.

discussed empyema and subdiaphragmatic abscess. On the 14th the cough was very troublesome, and in about half an hour a cupful of yellowish foul-smelling sputum was coughed up. The tenderness over the liver continued, but there was no pain. The other physical signs were unaltered. On the 15th a decrease in the dulness was noticed, and the voice-sounds were faintly heard over the dull area. From this date to the 22nd there was thought to be a further gradual improvement, the expectoration was less and the dyspnoea less marked. On the 20th an extension of the dulness behind towards the spines was noted, with distant tubular breathing and somewhat ægophonic voice-sounds. On the 27th there was more dyspnoea and much pain in the right side, which was relieved by linseed meal poultices. On the 28th the following treatment was ordered: Vin. Ipecac. $\text{m}x.$, Syr. Tolu. $\text{z}i.$, Quin. Sulph. gr. $ii.$, Acid Sulph Dil. $\text{m}v.$, Aq. Menth. Pip. ad $\text{z}j.$, 6tis horis, Guaiacol Carb. gr. $v.$, 6tis horis. The physical signs continued much the same and the sputum was still very foul.

On the 12th December the expectoration had become less, though still foul, and there was little or no alteration in the physical signs, and on the 13th the patient was allowed to get up after tea.

On the 23rd there was much dyspnoea and coughing, but very little expectoration. In the evening there was a rigor. Temperature 103° .

On the 24th the right side of the chest was explored in two places with a needle, but only blood was drawn off, which was sent up for bacteriological examination. The report received on the 27th was, "Coverslip preparation direct from the fluid show it to be practically pure blood, with perhaps a slight excess of leucocytes. A very few diplococci were found, which closely resembled the pneumococcus, but we were unable to demonstrate the presence of a capsule. Cultivations on agar and in broth remained sterile."

At this date the sputum was less in amount, but blood-stained and extremely foul. In addition to the physical signs previously noted there was a small patch of bronchial breathing high up on the right axilla, and loud bronchial breathing on the right side behind, over the lower part of the scapula, with ægophony and pectoriloquy.

On the evening of the 28th there was another rigor. Temperature 103.2° . On the 29th loud sonorous rhonchi were heard over the whole chest, and there was extreme tenderness for about an inch below the right costal margin and the inner half of the left costal margin. There was another rigor in the evening, and the temperature rose to 103° . He was a little delirious.

On the 31st Mr. Lane saw the patient and explored the chest with an exploring-needle in two places, but was unable to draw anything off. He noticed a feeling of hardness where the needle was inserted.

The tenderness over the liver still continued, and bronchial breathing with consonating râles was heard over the dull area on the right side. Rhonchi were heard all over the chest. Dr. Washbourn thought there was septic pneumonia.

During the remainder of his illness there was no marked alteration in the physical signs. The sputum was less in amount, but blood-stained. There were no rigors, and the tenderness over the liver became less marked. He gradually got weaker, and died on January 7th.

During the last week of his illness there was some diarrhoea, but previously to this he had been rather constipated.

Post-mortem (performed by Dr. Bryant), *No. 9. 1899.*—Sixteen hours after death.—Rigor mortis well marked. Considerable hypostasis. Body wasted. Brain not examined. About twenty ounces of thick greenish-coloured pus, mixed with blood, was found in the right pleural cavity and it was confined to the posterior part of the thorax. The posterior surface of the base of the right lower lobe was covered with a thick layer of ragged-looking, greyish-coloured lymph. An irregular-shaped opening was found in the posterior and lower portion of the right middle lobe communicating with the pleural cavity and with a good-sized cavity in the lung, the walls of which were irregular and ragged in appearance. This was evidently the tract along which the pus was evacuated during life. The posterior half of the left lower lobe was compressed and airless. The middle lobe was fibroid, the bronchial tubes were dilated, and it contained the above mentioned cavity which was filled with very foul-smelling pus. On the upper surface of the diaphragm on the right side a blood-clot about two inches in diameter was found; it appeared to have been caused by one of the explorations during life. Two exploration marks were also visible on the posterior surface of the right lung.

The heart weighed thirteen ounces. There was no pericarditis or endocarditis.

The stomach and intestine presented a normal appearance. The appendix vermiformis was normal. Some slightly turbid serous fluid was found in the peritoneal cavity between the diaphragm and the right lobe of the liver. The splenic vein was found to be full of thick greenish-coloured pus, the small veins forming it were nearly all filled with ante-mortem thrombi. The portal vein was filled with a soft, septic thrombus.

The liver weighed seventy-eight ounces. It was paler in colour than normal, and several small yellow slightly projecting areas could be seen on the upper and anterior surfaces of the right lobe, which on section proved to be a number of small abscesses. Throughout the liver the branches of the portal vein contained pus, and there were a large number of small pyæmic abscesses also.

Pancreas normal. Spleen ten ounces, normal. There was no lesion of any of the organs drained by the portal system to account for the portal pyæmia. At the time of the autopsy I suggested:—

(1). That a considerable amount of the pus coughed up from the empyema may have been swallowed, and that the pyogenic micro-organisms might have entered the portal system by absorption from the stomach and intestine.

(2). That the exploratory puncture which perforated the diaphragm might have inoculated the liver from the empyema.

Bacteriological examination.—A portion of the subdiaphragmatic fluid was obtained for bacteriological examination, all precautions been taken to prevent the possibility of any accidental contamination. No micro-organisms could be found by culture or by histological examination. A cover-glass preparation of some of the pus from the liver showed a large number of rod-shaped bacilli and some streptococci. Two distinct varieties were noticed, one short with rather pointed ends, the other long and thick with square end. Aerobic and anaerobic cultivations were taken. Long chains

of a long thick bacillus which appeared to be in pure culture were found in the aerobic broth culture at 37° C. No spores were seen. They did not retain the stain after being treated by Gram's method. In the anaerobic culture this same bacillus was found and also some streptococci (*vide* also Chart 2 at end of Paper).

CASE 3.—GALL-STONES.—SUPPURATIVE PYLEPHLEBITIS.—ABSCESSSES IN THE LIVER (LEFT LOBE).

Clinical, No. 562. 1896.

K. S., æt. 32, married woman, admitted into Clinical ward under the care of Dr. Taylor on October 29th, 1896, for pain in right hypochondrium and right shoulder, and for jaundice. There was a history of influenza six years ago and gastric ulcer three years ago. In March, 1895, she had an attack of pain in right side of abdomen and shoulder, without jaundice, which lasted a week. There were three succeeding similar attacks at intervals of six to eight weeks, and during the last a small stone was passed. About six weeks prior to admission she had another attack, but this time with jaundice.

The present attack commenced October 24th, with severe pain, which was treated with frequent injections of morphia. These had a dangerous effect, and patient nearly died. On October 28th she became jaundiced, and on October 29th she passed some blood.

Condition on admission.—Temperature 104·6°, respiration 32, pulse 140. She was thin, had an anxious expression, and was somewhat jaundiced. There are blisters produced by water bottles on both feet, and similar places on both hips.

Respiratory system.—A few râles at bases, otherwise normal.

Alimentary system.—Teeth covered with sordes. Tongue dry and covered with brown fur. Bowels opened on the day before admission. Liver somewhat enlarged. Gall-bladder not felt.

Eyes.—Conjunctivæ yellow. Pupils moderately dilated.

Progress.—On October 30th she had a good deal of pain and was more jaundiced. She was kept under morphia. On November the 1st and 2nd the motions were clay-coloured and the jaundice was very deep.

On November 3rd it was noticed that the spleen was easily palpable, and there was some aphthous ulceration of the posterior part of the hard palate and uvula. On November 4th blisters on right foot and left buttock showed signs of sloughing, and all the places were treated with creolin fomentations. Rigor at 6.30 p.m. On November 5th the patient was steadily getting worse, and there were some ulcers on the tongue in addition to those on the hard palate. Jaundice intense. Boracic fomentations applied. The slough on left foot is separating and exposing the tendon of the peroneus longus. On November 6th Mr. Symonds operated and found the gall-bladder empty, the cystic duct enormously dilated, and a large stone in it which was removed with some difficulty. Three other smaller stones were also removed. The fundus of the gall-bladder was fixed to a Paul's tube. Patient seemed a little better after the operation, and her pulse was good. On November 7th very little bile had come through the tube and she was unable to take her food because of the sores in her mouth. The urine contained less bile-pigment and the jaundice was slightly less intense (the condition of the urine is not mentioned before). Patient was put on nutrient enemata, 6tis horis. On November 8th she returned them and was fed by the œsophageal tube on beef-juice and milk.

November 9th. Patient died at 12 mid-day.

Autopsy (performed by Dr. Pitt), No. 463. 1896.—The body was emaciated and the skin jaundiced. There was lymph over the posterior surface of the right lung and at the apex. The lungs were cedematous and there was an early stage of pneumonia posteriorly in the left upper lobe with a definite line of demarcation. Some capsulated diplococci and other cocci were found in the lung-juice.

There was some early peritonitis with lymph on the surface of the spleen and liver.

The gall-bladder was securely attached to the edges of the abdominal wound and contained blood-clot. It was not distended, but the common and cystic ducts were much distended and the wall of the latter lacerated. A small pigmentary stone was found in the common duct. The main trunk of the portal vein was normal, but on splitting up the branches a recent thrombus was found partially obliterating the lumen of the lower branch to the left lobe. There was an abscess to the left of the gall-bladder containing greenish pus, and there were numerous small disseminated abscesses (none over 1 mm. diameter) in the portal areas of both lobes and apparently associated with the smaller bile-ducts. The liver was adherent to the diaphragm. The pancreas and spleen were normal, and the rest of the abdominal viscera were healthy (*vide* also Chart 3 at end of Paper).

CASE 4.—SUPPURATING GALL-BLADDER.—THROMBOSIS OF THE SMALLER BRANCHES OF THE PORTAL VEIN.

MR. LANE, No. 75. 1896.

E. W., æt. 60, married woman, formerly a laundress, was admitted under the care of Mr. Lane on March 1st, 1896, for biliary colic, with a view to operation. She had suffered from attacks of biliary colic for thirteen years, and stated that the first attack occurred after severe sea-sickness caused by a trip round the Isle of Wight. The attacks had increased in violence and frequency, and there had been (she said) forty or fifty attacks within the last year. (No description of an attack is given.) There was no pain between the attacks.

She had had eight children and several bad confinements, but no other illness. Her father was said to have died from a "tumour on the liver."

Condition on admission.—Pulse 100, respiration 24, temperature ?. She was well nourished and slightly jaundiced. No distension of the gall-bladder was detected on palpation, but there was tenderness on pressure in that region.

Urine.—1020 acid, trace of albumen, no sugar.

Progress.—March 2nd, operation 10 a.m.—An incision five inches long was made extending from the tip of the ninth rib vertically downwards on the right side. The gall-bladder was found firmly adherent to the transverse colon except at a point on the anterior surface towards the fundus. The bladder was opened, and several small stones the size of a pea, and one the size of a hazel-nut, were removed. No stones could be found in the ducts. The edges of the gall-bladder were sewn to the abdominal parietes, and a glass tube was tied in. The rest of the wound was then closed with deep sutures of salmon gut, and superficial sutures of horsehair.

A quarter of a grain of Morphine was given towards the end of the operation. Peptonised Milk, Nutrient Enemata, and Brandy were ordered.

Patient was sick at 2 p.m., 3.30 and 9.10 a.m. The vomit was greenish, and she complained of abdominal pain when she vomited. She was also sick six times during the night following. She had no pain except when sick. On March 3rd, the following day, at 10.15 a.m., she had a severe attack of abdominal pain, with flatulence, which she described as exactly similar to her previous attacks. There was a great deal of discharge from the wound, which was re-opened and again explored and found apparently healthy. She was sick again at 2 p.m., and had retching and flatulence at 4.15 p.m. At 7.45 p.m. she was in great pain, and at 9 p.m. another hypodermic injection was given. Her temperature, respiration and pulse-rates began to rise rapidly. At 1 a.m., March 4th, she was unconscious, and she died at 4.30 a.m. Shortly before death the temperature was 105.2°, pulse 150, respiration 36.

Autopsy (performed by Dr. Fawcett), No. 74. 1896.—The wound was perfectly healthy, and the gall-bladder firmly adherent to the incision in the abdominal wall.

The lower lobe of the right lung was compressed and airless, having apparently been rendered so by compression from below. There were old adhesions over left lung with a patch of collapse along the inner and posterior borders.

The heart weighed eleven and a half ounces and was healthy. There was no pericarditis, but there was about half a fluid ounce of clear fluid in the pericardial cavity.

The vessels generally were slightly atheromatous. The stomach and other parts of the alimentary canal were healthy.

The liver weighed forty-two ounces. The terminal branches of the portal vein lying in Glisson's capsule were found to be thrombosed, but no thrombi were found in the main trunk or larger divisions of the vein. The gall-bladder was one and a half inches long, and contained four small stones. It was firmly adherent to the lower surface of the liver and to the transverse colon, and on separating the adhesions a cavity containing about eight fluid ounces of pus was opened. The neck of the gall-bladder was much constricted, but below this constriction the duct was dilated and contained four small stones, and there was also a stone impacted in one of the branches of the hepatic duct. The spleen weighed six and a half ounces and was healthy. The kidneys weighed eight ounces and were granular. The cortex was thinner than normal.

There was an ovarian cyst the size of an orange attached to the broad ligament by a long pedicle.

Microscopic examination of the liver showed great increase of the interlobular connective tissue and small-celled infiltration (*vide* also Chart 4 at end of Paper).

CASE 5.—SUPPURATIVE PYLEPHLEBITIS.—MULTIPLE ABSCESSSES IN THE LIVER.—APPENDICITIS.—EMPYEMA.

Clinical, No. 480. 1895.

K. M., æt. 20, admitted into Clinical ward under the care of Dr. Pitt on September 16th, 1895, for pain in abdomen. About August 26th she had a sudden onset of pain in the abdomen, with vomiting and diarrhoea, causing much prostration. The vomiting ceased but the diarrhoea continued for three

days, and the temperature rose to between 101° to 103° in the evenings, falling to 99° in the mornings. The pain was referred to the umbilicus, with some tenderness in the right groin. Her medical attendant (Dr. Marshall) diagnosed the case as one of enterica. After about ten days' illness she began to have rigors and night sweating. About the eighteenth day her night temperature was 106° , and the rigors were severe. Dr. Pitt was called into consultation, and found signs of pleurisy, without effusion, in the right axilla, rigidity of the abdominal wall on the right side, and diagnosed appendicitis, with possibly hepatic abscess or general pyæmia.

Condition on admission.—Temperature 103.4° , respiration 28, pulse 144. Patient was much wasted, with an "abdominal expression" on her face. The tenderness of the abdomen was not marked, owing to a recent injection of morphia, but there was rigidity on the right side, from the groin to a line just above the umbilicus, with some impaired percussion resonance over the rigid area. No tumour could be felt, and there was no rigidity in the right loin. A rub could be heard in the right axilla. The diarrhoea had commenced again. There was a large bed-sore over the sacrum.

Mr. Symonds was called in, and operated at midnight. An incision was made in the right nipple line about opposite the umbilicus, and the liver and right kidney explored and found apparently healthy except that the edge of the liver reached an inch below the costal margin. The incision was continued to the groin, and the cæcum and appendix explored. The appendix was found firmly adherent to the cæcum, and the adhesions bled freely on attempting to separate them. The appendix was removed and a small quantity of pus, not more than *mxxx.*, found at its distal end. Iodoform was powdered on, and the wound packed with gauze. The patient bore the operation, which lasted ninety minutes, well. She was given morphia, gr. $\frac{1}{2}$, hypodermically, and ordered hot water to drink. Nutrient enemata 4tis horis. Rectum to be washed out with warm water twice in the twenty-four hours. On September the 19th and 20th she was taking fluid diet well, but pus was coming from the wound and patient was clamorous for morphia.

(No entries in report from September 21st to October 3rd.)

On October 3rd a rib was resected and a quantity of foul pus removed from the right pleural cavity, a puncture having been made previously and pus found. A finger was inserted and a small cavity felt above the liver. This was washed out with weak boric acid lotion and tubes were inserted. Patient was collapsed after the operation and a hot enema was given. She died on October 5th, at 6.30 a.m.

(The temperature varied between 99° and 100° in the mornings, and 101° to 102° in the evenings, rising on several occasions to 103° and once to 104° .)

Autopsy (performed by Dr. Pitt), *No. 380. 1895.*—The body was wasted and profoundly anæmic and there was a bed-sore over sacrum.

There was an empyema on the right side, extending from the third to the tenth rib, but limited to the region of the spine, and only three and a half inches across. This had not been reached by the axillary exploration. A portion of the ninth rib had been removed and there was an aperture in the diaphragm leading to an hepatic abscess. The posterior parts of both right lobes, especially the lower, were compressed. The lungs on the left side were mottled and there were a few ecchymoses on the posterior surface and two or three early patches of broncho-pneumonia.

There was lymph on both the upper and lower surfaces of the diaphragm on the right side.

A sinus in the right iliac fossa led to the stump of the appendix surrounded by a small collection of pus. The ligatures had slipped off the stump of the appendix, leaving the aperture patent.

The whole peritoneal cavity was bathed in turbid watery fluid, but there was no large collection, and infection appeared to have started from the surface of the liver.

There were several small abscesses on the surface of the liver and innumerable abscesses, mostly small, were exposed on section. An abscess one inch across was found in the duct, near the gall-bladder, which together with the bile-ducts contained pus.

There was a partially decolorized softening clot in the main trunk of the portal vein.

The pancreas contained numerous abscesses of considerable size, chiefly in the head, with brownish turbid contents.

The mucous membrane of stomach was ecchymosed and bile-stained (*vide* also Chart 5 at end of Paper).

CASE 6.—SUPPURATIVE PYLEPHLEBITIS.—PYÆMIC ABSCESSES IN THE LIVER.—APPENDICITIS.—PERFORATION OF THE APPENDIX.

Dr. Hale White, No. 201. 1894.

C. W. A., æt. 19, cabinet maker, was admitted into Stephen ward under the care of Dr. Hale White on 31st May, 1894, for pain in the right hypochondrium and pyrexia. On May 14th he was on the river at Oxford and got wet. On the following day he played tennis and went to the theatre. On the 16th he became suddenly ill with a rigor; he went to bed and vomited a quantity of bile and had great pain in the epigastric and hypochondriac regions.

From that date until admission he had rigors, as many as three a day, and there was always pain over the liver and occasionally in the right iliac fossa. No jaundice, no constipation, and no diarrhoea. He had lost flesh. Ten years ago patient had diphtheria and his throat has not felt quite right since. During his illness it has been very painful and sore.

Condition on admission.—Temperature 103·6°, respiration 26, pulse 80. A well-developed man. Has an anxious expression and looks extremely ill, anæmic, and collapsed.

Alimentary system.—Tongue dark and medicine stained. Slight erythematous rash over lower part of chest and abdomen. The abdomen moves slightly with respiration. There is fulness over the right hypochondrium and part of the epigastrium, and this area is painful and very tender, and the skin is hot and dry. There is a sense of resistance. The liver dulness reaches about half an inch below the costal margin, and above is limited by the sixth rib in front, angle of scapula and tenth rib behind. The dull area extends somewhat farther to the left than normal, encroaching on stomach resonance. There is tenderness and a feeling of resistance in the right iliac fossa, and some crepitation over an area the size of half-a-crown, but there is no loss of resonance.

Circulatory system.—Cardiac dulness normal. Reduplicated second sound at apex, otherwise normal. Pulse small, dicrotic and running.

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Respiratory system.—There is bulging on the right side of the chest below, with dulness and impaired entry of air. Above, the right side of the chest moves better than the left.

A diagnosis of abscess below the liver connected probably with old gastric trouble was made. Mr. Dunn was called in. June 1st. Rigor at 11.45 p.m. last night, and temperature 107°. Another rigor 10.30 this morning, temperature 99°.

Operation.—An incision two and a half inches long was made in the middle line above the umbilicus and abdomen explored. At first nothing abnormal was found, no fluid could be felt in chest through the diaphragm, but later, some foul-smelling pus welled up between the stomach and left lobe of liver. The cavity was irrigated, a drainage tube put in and the wound closed. There was another rigor after the operation, temperature 104°, and on the following morning there was a well-marked erythematous rash over the forehead, trunk, arms and legs.

June 2nd. An ounce of thick faecal-smelling pus was drawn up with a syringe last night, and about the same quantity this morning. Several rigors; rash almost gone. Urine acid, dark, 1024, no albumen and no sugar.

June 3rd. More pus from wound; no rigors.

June 4th. Patient much worse. Rash has appeared again and there is more foul pus from the wound. Pulse very rapid and thready. Patient died 7.45 p.m. Just before death a quantity of brownish fluid containing "coffee ground" material was vomited.

Autopsy (performed by Dr. Bryant), No. 220. 1894.—The body was well nourished, and there was no anasarca. There was an incision in the median abdominal line three inches long, commencing two inches below tip of ensiform, sutured with gut, and with an opening left in which was a drainage tube.

There were a number of sub-pleural petechiæ on the surface of both lungs, and about five to six fluid ounces of turbid fluid were found in the right pleural cavity. There was a little compression of the right base, and the lungs were injected.

The heart weighed nine ounces. There was some P.M. staining of the endocardium and aorta.

There were about thirty fluid ounces of turbid sero-purulent fluid in the peritoneal cavity, and the peritoneum had flakes of yellow lymph adhering to it.

There was a tract leading from the incision to the hepatic flexure of the colon where the drainage tube had rested.

The end of the vermiform appendix was found extending upwards under the lower border of the third part of the duodenum. It was the seat of an abscess cavity (1½ inch by ¾ inch by ¾ inch) containing foul-smelling pus. There were two small communications with the peritoneal cavity, and also a direct communication with the mesenteric vein which was filled with a septic thrombus extending into the portal vein and its larger branches.

There were a large number of abscesses in the liver, the left lobe was most affected, and the branches of the portal vein contained septic thrombus. The liver was not weighed. Behind the cæcum was a small abscess which had been caused by a perforation of that viscus. The kidneys weighed ten ounces and were healthy. The spleen weighed eight ounces and was soft. The other abdominal viscera were healthy (*vide* also Chart 6 at end of Paper).

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CASE 7.—SUPPURATIVE PYLEPHLEBITIS.—GASTRIC ULCERS.—PYÆMIC
ABSCESSSES IN THE LIVER.

Dr. Goodhart, No. 272. 1891.

A. M., æt. 19, a carman, was admitted into Philip ward under the care of Dr. Goodhart on July 28th, 1891, for diarrhœa and abdominal pain. On July the 19th he had eaten some haddock. Afterwards while sitting down he suddenly felt acute pain in the pit of his stomach and a sensation as if something had given way. He was assisted home and put to bed. The pain continued till the following morning, and was intense and accompanied by vomiting, the vomit being bile-stained and containing a little blood. In the evening the pain recommenced. On July 21st an enema was given and a motion passed, being the first since the onset of his illness. From the 21st until admission he had continuous pain and diarrhœa, the pain being slightly relieved by the passage of motions. There has been no blood noticed in the stools. He has been feverish and slept little, but had no delirium. His abdomen has been much distended and he has passed a large quantity of flatus. No spots on the abdomen.

Condition on admission.—Temperature 99·8°, pulse 92.

Alimentary system.—Lips dry—covered with sordes. Mouth dry. Tongue red at the edges and the tip, furred on the dorsum; abdomen tense—extremely tender, tension most marked in flanks and between umbilicus and pubes, flaccid and incurved between umbilicus and sternum. There is dulness where the tension is most marked, viz., hypogastric, right and left inguinal and left lumbar and left hypochondriac regions. In the right lumbar region the resonance is diminished but there is not absolute dulness. Patient lies on his back with his legs fully extended. Abdomen is twenty-eight and a half inches in circumference at the umbilicus, thirty-one and a half at anterior superior spines. Liver dulness extends from fifth rib in nipple line to just below costal margin.

A motion was passed consisting of some well-formed light-coloured fæces and a fluid portion containing mucus and a few clots of red blood. The pain was relieved by defæcation and the act itself was painless.

Respiratory system.—Breathing chiefly thoracic but the upper part of the abdomen moved slightly. T. V. F. impaired below sixth rib on each side. A few râles heard at the left base.

Circulatory system.—Impulse in fourth left space, half an inch internal to the nipple. First sound accentuated.

Urine.—Sp. gr. 1018, acid, no albumen, no sugar, no blood.

July 30th. Patient has had little sleep owing to great pain. Two motions passed with no blood.

July 31st. Pain less; abdomen smaller and less tense.

August 1st. Rhonchi on left side of chest in front, well marked at apex. Pain and tenderness diminishing. Right inguinal region most tender.

August 4th. Tenderness only found now just below the umbilicus and in the right inguinal region. Motions loose and bloodless.

August 5th. Three rigors, with temperature between 104° and 105°.

August 6th. Patient exhausted. Pulse and respiration rapid and weak.

August 8th. Patient vomited eight times during the night, the vomit being greenish in colour. Profuse sweating; loose motions; another rigor.

August 10th. Patient very collapsed. Mr. Lucas saw the patient and it was decided to operate. An incision was made between the umbilicus and pubes and a condition of general suppurative peritonitis found. About eight ounces of foetid pus were let out and a drainage tube put in. The patient was very exhausted and died soon after the operation.

Autopsy (performed by Dr. Bryant), No. 307. 1891.—The body was much emaciated.

The lungs and heart were healthy.

The stomach was distended and adherent to the under surface of the liver. There was a longitudinal ulcer two and three-quarters by three-eighths of an inch along the middle of the lesser curvature, and anterior to and to the right of this ulcer two smaller ulcers, one the size of a sixpence the other the size of a threepenny-piece. Both of the smaller ulcers had perforated but had become adherent to the under surface of the left lobe. Between the large longitudinal ulcer and the lesser, and also burrowing into the under-surface of the left lobe of the liver, was an abscess containing about two fluid ounces of foul pus.

The intestines were covered with lymph and pus and were matted together.

There was a considerable amount of pus in the peritoneal cavity.

The liver weighed sixty-eight ounces. There were several small abscesses in the right lobe, some of them superficial, and there was pus between the diaphragm and the liver. There was also the large abscess on the under-surface of the left lobe mentioned above.

The portal canals were full of foul pus.

The spleen weighed seven ounces. On the middle of the upper surface there was an abscess containing about one ounce of pus with several smaller abscesses around. There was lymph and pus on the capsule (*vide* also Chart 7 at end of Paper).

CASE 8.—MULTIPLE HEPATIC ABSCESSSES.—SUPPURATIVE PYLEPHLEBITIS.— ULCERATION OF THE RECTUM.

Dr. Goodhart, No. 322. 1890.

E. D., æt. 49, married woman, was admitted into Mary ward under the care of Dr. Goodhart on the 13th of September, 1890, for jaundice. She had been married twenty-nine years and had had three or four miscarriages, no children living. She had always lived well, drank beer but no spirits, and had enjoyed good health, except for habitual constipation and bilious attacks, during which, however, she had not hitherto been jaundiced. The family history was good. Her present illness began two months ago, with an attack of vomiting in the morning. The vomited matter was green, and she had a rigor and felt like "ice." She went to bed and remained there till admission. A second attack came on while in bed, and she turned yellow. Her urine has been very dark in colour, and her motions at first were white and then yellow. On admission, her bowels had not been opened for two days. Latterly she had not been sick.

Condition on admission.—Temperature ?, pulse ?, respiration ?. Patient seemed very weak ; skin yellow, lips pale, sclerotics jaundiced.

Alimentary system.—Tongue red and clean, mouth dry, and patient complained of thirst. Abdomen rather resistant. Liver dulness reached just below costal margin. Spleen felt with difficulty. A sharp aching pain across abdomen.

Circulatory system.—Cardiac dulness and sounds normal.

Respiratory system normal.

Patient said to have lost flesh a great deal lately.

Urine 1016, acid, dark coloured, no albumen, gives Gmelin's test for bile pigment.

Progress and treatment.—Patient was given M.M. cum M.S. ʒj. t.d.s., Acid. Hydrocyan. dil. miiij., M. Effervescent. ad ʒj., and ordered a diet of fluids.

September 16th. Not quite so yellow, no pain, only a slight uneasiness.

Conf. Sennæ et Sulph. ʒj. statim.

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|---------|-------|---|
| " | 18th. | Slight diarrhœa, with pain across abdomen. |
| " | 20th. | Slight diarrhœa. Takes food badly. |
| " | 21st. | Bowels opened twice. Second motion a steel-grey colour but well formed. |
| " | 25th. | Was sick after eating an egg for breakfast. No pain. |
| " | 27th. | Much retching but no vomiting. Cannot take food. |
| " | 29th. | Sickness and diarrhœa. <i>Marked bulging noticed on right side of chest.</i> Morning temperature 96·8°, evening temperature 98°. |
| October | 2nd. | Diarrhœa continues. Continual retching. Occasional vomiting. Morning temperature 96·8°, pulse 120, respiration 30; evening temperature 100·8°, pulse 120, respiration 24. |
| " | 6th. | A large clot of blood passed per rectum. |
| " | 7th. | Urine, sp. gr. 1020, dark brown, gives Gmelin's and Pettenkofer's tests. Slight œdema of legs. |
| " | 8th. | A few small clots of blood passed. Morning temperature 96·2°, pulse 128, respiration 24; evening temperature 99·0°, pulse 120, respiration 28. |
| " | 9th. | Dr. Goodhart observed that bile in the motions, without diminished jaundice, pointed to a collection of small abscesses in the liver. |
| " | 10th. | Motions tested for bile. Gmelin's reaction obtained. More clots per rectum. The rectum was examined and a small cavity, half an inch in depth, with smooth walls, not everted, and a hard smooth base was found. The examination was not painful. Morning temperature 97°, pulse 128, respiration 24; evening temperature 98·4°, pulse 120, respiration 28. |
| " | 13th. | Motions greenish yellow, with small blood clots present. Another rectal examination made and another ulcer discovered on the anterior wall of the rectum. |

Drs. Goodhart and Shaw thought they were not malignant. Intestinal obstruction and pylephlebitis were diagnosed. More distinct resistance in the right hypochondrium and a lump felt on deep pressure.

October 14th. Patient died at 2 p.m.

(N.B.—Highest temperatures after September 28th were: September 30th, 99·8°, October 2nd, 100·8°, October 5th, 100·2°. Temperature after September 28th was generally subnormal.)

Autopsy (performed by Dr. Pitt), 385. 1890.—The body was very thin and deeply jaundiced as to the skin and conjunctivæ.

The lungs were jaundiced and there was slight œdema of the right base but they were otherwise normal.

The heart weighed seven and a half ounces and was healthy.

The stomach was healthy, and the duodenum normal as far as the biliary papilla, which was surrounded by a villous growth about the size of a walnut, that may have somewhat obstructed it. The growth appeared to be innocent, for there was no thickening, and apparently no secondary deposits.

The remainder of the small bowel was healthy and so was the large intestine as far as the rectum. Three inches above the anus were two circular ulcers, one on the posterior wall and the other on the right lateral wall, each about the size of a half-crown piece. Their edges were undermined and their bases, formed by the muscular coats, were sloughy.

No thrombi could be seen in the mesenteric veins.

The liver weighed forty-nine ounces. It was deeply jaundiced and very soft. On section, pus exuded from the various points, apparently from the smaller branches of the portal vein, and there were also actual collections of pus in the liver-substance.

No thrombi could be found in the main branches of the portal and hepatic veins.

Microscopically the liver tissue was extremely fatty. There were several collections of small round cells replacing the liver cells, but no connection could be made out between these collections and the branches of the portal vein.

The gall-bladder was healthy.

The spleen weighed five ounces, and was soft.

The kidneys weighed eleven and a half ounces, and were soft, but otherwise healthy.

The external os uteri was stenosed from cicatrization, but would admit the end of a probe. The cervical canal was small and full of glairy fluid.

The ovaries were small and fibrous, and about the left tube and ovary there were old adhesions (*vide* also Chart 8 at end of Paper).

CASE 9.—SUPPURATIVE PYLEPHLEBITIS.—MULTIPLE HEPATIC ABSCESSES.—APPENDICITIS.

CLINICAL, No. 355. 1890.

M. H., æt. 16, domestic servant, was admitted into Clinical ward under the care of Dr. Goodhart on March 15th, 1890, for pyrexia. Her illness commenced on March 7th with bilious vomiting and constipation, and she was ordered M.M. cum M.S. by her doctor, which relieved her. On the 9th the vomiting returned, and she was again constipated. Calomel gr. iii. followed by M.M. cum M.S. was prescribed, and at midnight she had acute abdominal pain.

On the 10th she had much pain over the ileo-cæcal region, increased by pressure. There was some tympanites, and a dry, furred tongue. Temperature 104°, pulse 126, small and feeble. In the evening the symptoms were more marked, temperature 104.4°, pulse 128. The treatment ordered was Pulv. Opii. gr. ½, Ext. Belladon. gr. ¼, 4tis. horis, with Mist. Pot. Cit. and Tinct. Aconiti. miii. On the 11th she was very restless and delirious, and there was retention of the urine for which a catheter was passed. On the 12th there was much tympanites, and the bowels were opened for the first time since

the 9th. A single scybalous mass was passed, followed by dark, watery, offensive evacuations containing undigested milk. The treatment was changed for Mist. Pot. Cit. with Liq. Bismuthi mxx . 4tis. horis. On the 13th there were several liquid motions and the other symptoms were better; there was much pain and tenderness over the transverse colon. On the 14th, four liquid motions, no tympanites. She had influenza about one month before the commencement of present illness.

Condition on admission (March 15th).—Temperature 102.4° , pulse 112, respiration 36. Abdomen full and resistant. Pain and tenderness, chiefly across the upper part of the abdomen, complained of, also pain in the back. Spleen not felt, and no increase of splenic dulness. No rash. Tongue covered with white fur. No headache, and no history of headache.

Respiratory System.—Normal.

Circulatory system.—Heart normal, face flushed, pulse full and bounding.

Urine.—Sp. gr. 1025 acid, no albumen; urates present.

On the 17th an enema was given, and a motion of well-formed normal faeces passed. At night her temperature rose to 105° , and she was sponged.

On the 18th temperature was down and patient comfortable. In the evening her temperature rose to 103.8° , and she was again sponged.

On the 19th the temperature was down to 99° , pulse 112, dicrotic: a formed motion not typhoid in character was passed. Two (?) typhoid spots on abdomen.

On the 22nd abdomen was about the same, tenderness chiefly in the right hypochondrium. No more spots seen, but a motion somewhat typhoid in character passed. Temperature low in the morning and up in the evening; marked rigor lasting some time when the temperature is high.

On the 24th an indurated lump was felt in the right iliac region. More rigors.

On the 27th Dr. Horrocks examined patient and reported the hymen perforated. The uterus felt undeveloped and was to the left side. No effusion into the broad ligaments. No tenderness.

On the 28th Mr. Lane operated under an anæsthetic (CHCl_3). A well-defined oval tumour was felt in the right iliac fossa. An abscess containing very foul pus was found extending along the outer side of the cæcum for about an inch and a half, surrounded by adhesions. The appendix was separated and removed, the cavity washed out with Lot. Hyd. Perchlor. (1 in 2000) and a drainage tube inserted.

After the operation patient was kept under Morphia and fed on nutrient enemata; nothing was given by mouth. There was a good deal of foul discharge following the operation.

March 30th. There was a pleuritic rub on each side.

" 31st. There was a rigor. Temperature 102° rising next morning to 103° .

April 1st. Pain over lower part of abdomen.

" 2nd. Rigor. Temperature 102° .

" 3rd. Rigor. Temperature 103° . Tenderness in right hypochondrium and right lumbar regions, also in right iliac region. Slight redness over right hypochondrium.

" 4th. No rub heard now. Patient sleeps well. Wound looks healthy. Motions normal. Temperature 98.4° to 101.8° , pulse 120 to 130, respiration 26 to 28

- April 7th. A small abscess over the base of the sacrum was opened. Still pain in right iliac region. Free discharge from wound.
- " 8th. Rigor. Temperature 104°.
- " 10th. Slight tenderness over liver.
- " 17th. Vomiting and retching several times. Abdomen full and tender in the right iliac fossa.
- " 20th. Tympanites.
- " 21st. Purpuric eruption in the epigastric region. Temperature 102°, pulse 130. No change in the wound.
- " 23rd. Some fresh purpuric spots in the umbilical region. Tympanites not so marked. Diarrhoea.
- " 24th and 25th. Diarrhoea.
- " 26th to 28th. Patient sank gradually and died at 10 p.m. on April 28th.

Autopsy (performed by Dr. Pitt), No. 149. 1890.—The body was much emaciated. There were loose flocculent adhesions over right pleura, but none of very recent date.

In the posterior-inferior part of the right lung there was a wedge-shaped area containing scattered abscesses with surrounding imperfect pneumonia, but the tissue was not solid, and the limits were ill-defined.

The heart was very small.

There was an old sinus in the right iliac region which was dry and free from pus.

On opening the abdomen some adhesions were found, and on cutting through the omentum about one ounce of foul watery pus was let out. There was a collection of about the same quantity in the pelvis.

The cæcum looked healthy, and the stump of the vermiform appendix was closed.

There was a suppurating thrombus in the superior mesenteric vein which could be traced into a mass of purulent material around it. The branches of the portal vein were filled with pus, and the main trunk was obliterated by a firm thrombus.

In the right lobe of the liver were numerous abscesses, most of them small and collected into groups. There were only a few abscesses in the left lobe. In one or two of the abscesses there was communication with a bile-duct.

There were flocculent adhesions over the liver and spleen. The rest of the viscera were healthy (*vide* also Chart 9 at end of Paper).

CASE 10.—SUPPURATIVE PYLEPHLEBITIS.—MULTIPLE ABSCESSES IN LIVER.—

PYOSALPINX.—SUPPURATING OVARY AND PELVIC ABSCESSES.

Mr. Howse, No. 117. 1886.

A. E. H., æt. 20, a teacher, was admitted into Charity ward under the care of Mr. Howse on February 4th, 1886, for abdominal tumour. On January 15th, she had some pain in the abdomen and took some castor oil, which made her sick. The pain continued on and off, and on January 22nd she took some more castor oil after which she again vomited. In the evening of the same day she noticed a swelling in her abdomen.

Her previous illnesses had been measles and whooping cough in childhood, and she was said to have had inflammation of the ascending colon eighteen

months ago, which confined her to her bed for a month. She had another similar attack in October last. She was not married. The family history was good.

Condition on admission.—Pulse ?, respiration ?, temperature ?. She appeared healthy-looking with a slight flush on her cheeks, but felt weak from lying in bed. A tumour, one and three-quarter inches in diameter, was seen on the right side of the abdomen in the line of the right rectus muscle and about midway between the costal margin and the umbilicus. The tumour was tender and fluctuating and was felt to extend obliquely upwards and to the left.

Progress and treatment.—Milk, beef-tea, and farinaceous diet were ordered. On February 8th the tumour was slightly larger and more raised, and the patient had been in pain all night. The urine was examined and found slightly acid, sp. gr. 1026, no albumen, no blood. A few pus corpuscles were seen microscopically.

On February 10th the skin was reddened over the tumour. It was increasing in size towards the right hypochondriac region and measured three and a half inches in diameter.

On February 14th chloroform was given and the tumour incised. About 3vj. of pus escaped which had a strong fæcal odour, and it was therefore thought to come from the transverse colon. There was a good deal of venous hæmorrhage after the pus had come away. A drainage tube was put in and the wound dressed with carbolic gauze. There was some sickness after the operation, and the temperature was 98° and pulse 88.

Mist. Bism. et Morphinae 6tis horis was ordered, and milk, ice and soda water.

On the 16th the urine was dark brown in colour, acid reaction, sp. gr. 1028, pink deposit of urates; no albumen, no blood, no bile-pigment, no indican.

On the 19th chops were ordered.

March 1st. Stout.

" 3rd. Chicken.

" 7th. Liq. Hyd. Perchlor m℥xxx.

Mist. Ac. Hydrochlor. ʒi. t.d.s.

The wound was being dressed every few days. It continued to discharge, but no symptoms are mentioned.

On March 24th, 28th and 30th. Tannic Acid was sprinkled on to repress exuberant granulations.

On April 1st, Dr. Hale White examined the patient and found slight dulness at the right apex, with bronchophony, prolonged expiration, and almost tubular breathing. He thought there was phthisis at the right apex.

On April 10th she was sick during the night and again in the morning. On the 11th and 12th the sickness continued, and there was great abdominal pain felt, especially when breathing. Her face was very white and drawn and her lips dry. Urine acid, sp. gr. 1025, no albumen, no sugar, much deposit. She continued very restless, but the pain was easier, and there was no more vomiting. She died on April 14th at 8.30 a.m. No rigors are mentioned throughout.

Autopsy (performed by Dr. Goodhart), 127. 1886.—The body was very spare.

There was a partly healed wound in the epigastrium, with a drainage tube inserted.

There was acute pleurisy on the right side of the chest, with much yellow lymph.

There was some collapse at the base of the right lung, but the lungs were otherwise healthy.

The heart weighed six and a quarter ounces and was healthy.

There was a caseous gland in the mediastinum.

There was a large quantity of pus in the peritoneal cavity, but this had no connection with the superficial abscess opened and drained externally.

The liver was adherent to the diaphragm, beneath the superficial abscess, and at this point (between the gall-bladder and the round ligament), was a cluster of small points of pus having the distribution of a small branch of the portal vein.

Just beyond the bifurcation of the portal vein about $\frac{3}{4}$ in. of pus were found, and the wall of the vein was shaggy from adherent lymph. The gall-bladder and other parts of the liver were healthy.

The pelvis was for the most part filled with a globular mass, which was fixed by tough inflammatory adhesions to the pelvic wall, and to which the intestines were adherent. This proved to be the right ovary, which on section showed many caseous or suppurating points, and which was surrounded by a sinuous abscess, which burrowed about among the adherent coils of intestine and finally opened near the cæcum, simulating perityphlitis. The left ovary was enclosed in a tough coat of fibrous tissue.

The Fallopian tubes were both adherent and full of pus.

The uterus and vagina were healthy.

The cæcum and appendix were themselves quite healthy.

There was a caseous lumbar gland on the right side.

The pelvis of one kidney was dilated.

The other viscera appeared healthy (*vide* also Chart 10 at end of Paper).

* CASE 11.—SUPPURATIVE PYLEPHLEBITIS.—MULTIPLE HEPATIC ABSCESES.
—APPENDICITIS.—PELVIC ABSCESS.—ULCERATION OF THE COLON.

MR. HOWSE, No. 180. 1880.

W. G. H., æt. 18, brushmaker's assistant, was admitted into Naaman ward under the care of Mr. Howse on September 17th, 1880, for discharging sinuses in left side and left groin. About 20th May he had pain above the left groin, and hard lumps appeared both in the groin and in his left side. Linseed poultices were applied and the lumps in the side burst two days later by two small openings. Five days later the abscesses in the groin burst.

About the middle of June he had shooting pains in his left side. He had jaundice three or four times during his illness, the last time being about the middle of July. Family history good. No previous illness.

Condition on admission.—Temperature 100°. Thin, pale, anæmic; face has a yellowish pallor; pearly conjunctivæ. On the left side, an inch and a half above the middle of the iliac crest is a swelling four and a half inches by two and a half inches, with a small opening discharging foul green pus very freely. About half-way between this swelling and the left anterior superior spine there is another fluctuating swelling about the same size with surrounding œdema. In the left groin there is a triangular opening discharging greenish pus. *Urine* acid, no albumen.

September 18th. Morning temperature 99·8°; evening temperature 98·8°. Both the swellings in the side were opened under CHCl_3 . There was a free

* This case is also reported by Dr. Carrington in his paper.

discharge of pus from both wounds and they were found to communicate. A probe could be passed forward in the direction of the opening in the groin. No communication with the spine could be positively made out. A large drainage tube was inserted. There was a great deal of discharge following the operation, and patient was much easier.

- September 28th. Patient complained of stiffness in his back on sitting up, and the lower dorsal region was tender.
- October 4th. Shooting pains in left side. Unable to lie on his back because of the pain so caused.
- " 8th. No discharge from the wound, and the drainage tube was removed.
- " 10th. Wound dressed with resin dust.
- " 18th & 19th. Large quantity of urates present in urine; no albumen.
- " 20th. Fluctuation above the crest of the ilium.
- " 23rd. Urine, sp. gr. 1032; sugar present.
- " 24th. The abscess above the iliac crest was opened and a large quantity of pus evacuated. A probe could be pushed down almost to the groin.
- " 28th. Urine tested every day since 23rd, but no more sugar found.
- " 29th.)
- November 2nd.) Albumen present in urine.
- " 3rd.)
- " 6th. Shooting pain in the left side on breathing.
- " 9th. Pain has disappeared. Not much discharge from the sinuses.
- " 29th. A large quantity of albumen in the urine.
- December 29th. Patient complains of a feeling of fulness and tension in the left (? right) groin.
- January 5th. Mr. Howse examined the swelling in the right groin and diagnosed abscess. He said this confirmed the diagnosis of disease of the spine.
- " 15th. The abscess in the right groin was opened under chloroform and a large quantity of blood and pus evacuated.
- " 22nd. Patient complains of shivering in the morning and subsequent sweating. The bowels have not acted for six days.
- " 23rd. A double Bryant splint was applied from the feet to the axillæ. Patient feels pain in the muscles of his knee, which had remained flexed for a long time. Morning temperature 98°; evening temperature 98°.
- February 5th. The patient is used to the splint after suffering at first great pain. Evening temperature 99°.
- " 20th. A large abscess appeared on the left side, near the iliac crest, but was not opened, by request of patient's mother. Morning temperature 100·3°.
- " 24th. An abscess opened on left side, just below ribs, from which fæcal matter escaped. Patient in great agony and never at rest except when under morphia.

		Morning temperature 100°. Morphine gr. $\frac{1}{4}$, hypodermically. Urine 1015, neutral; no albumen or sugar.
March	3rd.	Much pain in the back. Morning temperature 99°; evening temperature 101°.
"	10th.	Abdomen slightly distended and tense, tender on palpation. The patient lies with his legs drawn up. Flattening, diminished resonance and tubular breathing noticed over the right apex. The back and centre of the tongue was coated with brown fur, the sides were clean. Morning temperature 102.4°, pulse 140, respiration 35.
"	12th.	Died 4 p.m.

There is no temperature chart in the report. The temperature had been taken and reported occasionally. The highest records are, March 7th, morning temperature 101.8°; March 9th, morning temperature 104°; evening temperature 103°; March 11th, morning temperature 102°; evening temperature 103.6°. Most of the other temperatures recorded vary between 98° and 100°. The pulse and respiration rates have not been recorded.

Autopsy (performed by Dr. Goodhart) No. 75. 1881.—The body was much emaciated, and there was slight oedema about the ankles.

There was a sinus in the right groin which ran inwards and opened into an abscess behind the pubes, and three sinuses in the left groin. Two had all but closed and the third opened into the descending colon.

There was about half an ounce of pus in the right pleural cavity and the pleura was covered with yellow lymph.

The greater part of the lower lobe of the right lung was solid, and at the hinder part of its lower border was a brownish patch of softening.

There were several small infarctions in this lobe, and it was noticed that the lymphatic vessels were much distended, some with pus and others with inflammatory material, and that the lymphatic glands on this side of the chest were much enlarged.

The diaphragm was adherent to the lung above and to the liver below.

The heart weighed seven ounces, and the wall of the left ventricle was disproportionately thick. It was otherwise healthy.

The vena azygos was unusually large and distended.

In the abdomen there were many adhesions of a somewhat chronic kind, those between the liver and diaphragm being especially tough, with pus imprisoned in the loculi formed by the adhesions.

In the lower part of the abdomen behind the pubes and between it and the coils of small intestine was an abscess cavity with thickened and much discoloured walls. This abscess extended to and surrounded the cæcal appendix, and had burrowed down into Douglas' pouch and opened into the rectum some two inches from the anus. It also extended to the left side of the abdomen and had burrowed up beside the descending colon.

The sinus in the right groin communicated with it behind the pubes.

The intestines were found to be lardaceous in places, and two small openings were found in the ileum about two inches from the ileo-cæcal valve and were thought to be due to extension from without inwards of the pelvic abscess.

The appendix was long, and a small opening was found near its end, into which a probe passed for about 2 mm. but no communication was found with the canal of the appendix.

The mucous surface was puckered and discoloured, but no perforation was found. The colon was healthy as far as the splenic flexure, and here was a sinus leading to the groin and several ulcers opening into thickened and discoloured mesentery and in close proximity to the upward extension of the pelvic abscess. There were also the scars of healed ulcers.

The liver weighed fifty-nine ounces. Several adhesions were seen on the surface which on section proved to be the scars of old abscesses, with in some instances a small yellow central deposit. One abscess was in the caseous state without any scar tissue. There were also numerous points of gray cirrhotic-looking material with two or three central specks of yellow which looked like early caseous degeneration of fibroid matter. From some of the points pus was coming, and they were all in the portal canals.

The portal vein in the portal fissure was completely blocked by a soft clot which contained yellow pus in its centre. Some large veins were found in the portal fissure in front of the portal vein, which probably communicated with the inferior vena cava and so relieved the circulation.

This plexus of veins was afterwards partially dissected out and some were found to run forwards into the round ligament, and others backwards to the diaphragmatic opening of the vena cava, where they became lost.

The œsophageal veins were not dilated. It was impossible to make out clearly the whole of the collateral circulation; but in addition to what has been noted, the left spermatic vein was found unduly large.

The main bile-ducts were healthy and the gall-bladder contained healthy bile, but a further examination showed that the bile-ducts in various parts of the liver were implicated, and also that the fibrous tissue round the portal vein had caused pressure upon the ducts, which were dilated, and in some of the smaller ones the bile was inspissated.

The spleen was a good specimen of the sago spleen of lardaceous disease. Its vein was plugged and full of pus, and there was an abscess in the splenic tissue.

Microscopical examination of the liver showed an immense amount of new fibrous tissue in the scar-like patches,—an advanced form of cirrhosis.

The case was thought to be one of primary ulceration of appendix giving rise to obstruction of the portal vein and pylephlebitis and ulceration of colon.

Remarks on the above case in the Path. Soc. Trans., 1881, Vol. xxxii., p. 137, by J. F. Goodhart, M.D.

The patient was a boy, æt. 18, who was admitted into Guy's Hospital under my colleague, Mr. Howse, for an abscess of obscure origin in the left groin. This abscess communicated with the descending colon. He was ill about ten months in all, and died worn out by emaciation, suppuration, and lardaceous disease of the viscera.

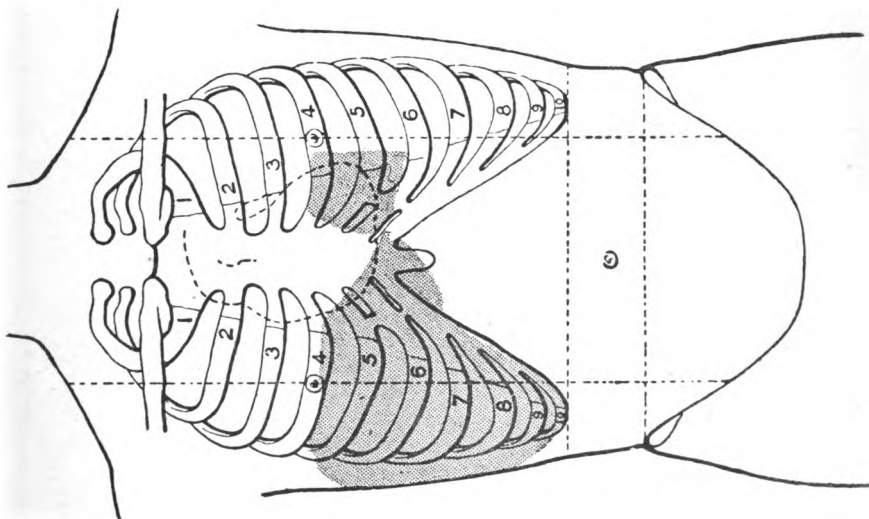
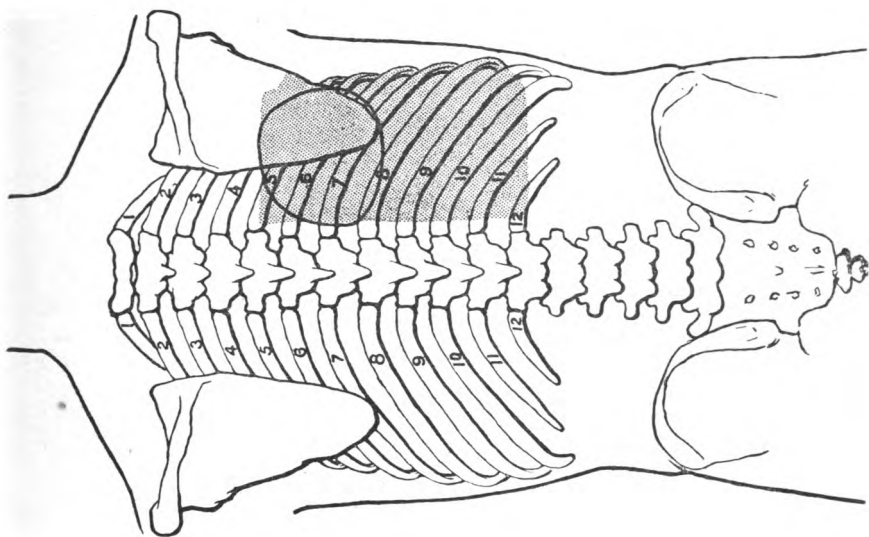
At the inspection a pelvic abscess was found, which had burrowed about in various directions forming sinuses in each groin and also in the left loin, the latter opening also into the colon. The abscess had opened also into the general peritoneal cavity and set up suppurative peritonitis. The colon was ulcerated and there were appearances in the cæcal appendix which looked like the scar of an old ulcer. For this reason, because no other probable source of abscess was to be found, I concluded that typhlitis had possibly

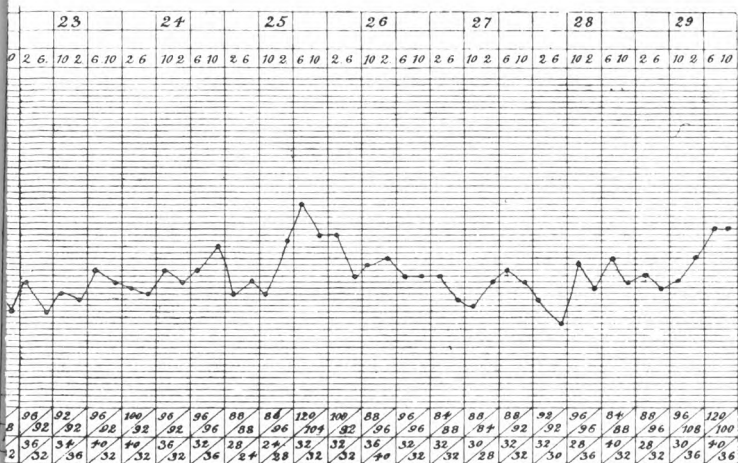
been the cause of all the mischief. The left pleura was the seat of a local empyema; there were infarctions in the lung and a lardaceous spleen and intestines. The state of the liver is also particularly worthy of record. The portal vein was completely occluded, its walls much thickened, and containing in its centre purulent matter tinged with bile-pigment. The greater part of the liver was perfectly healthy, but in various parts scars were found which mostly had a caseous centre and in one spot near the surface was a distinct mass of caseous matter, apparently an old or residual abscess.

Under the microscope a quantity of new fibrous tissue was seen to have destroyed the liver substance almost completely at the spots described as scars.

The interest of the case seems to hinge upon the probability that the suppurative pylephlebitis had ended, not as usual in the death of the patient, but in cicatrization, and in doing so had set up isolated centres of cirrhosis such as have been described by Hanot, Charcot and others in connection with the ducts. A condition similar to this has been observed, and such cases have been recorded by Dr. Moxon and myself, to exist occasionally in the kidney.

CASE I.





ON SOME POINTS IN THE TREATMENT OF ACUTE INFANTILE DIARRHŒA. SOME HINTS FOR THE YOUNG PRACTITIONER.

By G. E. HALSTEAD, M.D.

If a general practitioner looks over the retained portion of his old books of death-certificates, he will find that the vast majority of the fatalities come under a very few headings. Heart disease, phthisis, cancer, pneumonia, appendicitis, old age with its degenerate arteries and heart, and, in children, broncho-pneumonia in winter, diarrhœa in summer, and convulsions all the year round (cause often undetermined), with an occasional specific fever cause nearly all the deaths. Some diseases, such as aneurism and acute Bright's disease and chronic spinal cord lesions, which loom so large in hospital work, are not common in general practice. For purposes of examination, students have to try to learn a good deal about everything. They must be able to write as much about the vagaries of Raynaud's disease as about diseases of the stomach, although knowledge of the latter will subsequently prove to them much more useful.

Amongst the diseases which are of the greatest importance in general practice, perhaps there are none more important than the following three:—pneumonia, appendicitis, and acute infantile diarrhœa. When one considers their frequency, their fatality, and the great shock caused to the patient's friends if, as often happens, an apparently healthy being dies within a week or so,

there can hardly be many other diseases of which it is so important for the medical man to know thoroughly all the details and the best treatment.

The present paper is only concerned with some points in the treatment by diet and drugs of acute infantile diarrhœa during the first week of the illness. In every large town diarrhœa slays its hundreds of babies in August, and its treatment is unsatisfactory from every point of view. What follows is only written for students qualifying or recently qualified who have probably hardly had the opportunity of watching carefully from day to day, and the responsibility of treating from start to finish, a single case of this kind. Most men who have been in practice for some years have accustomed themselves to the annual death-certificates, and have probably sunk into a groove of treatment out of which they are only jolted when one of their own babies is at death's door with the complaint. And the consultant cannot be of much use in these cases unless he sees the baby daily with us, for the handle of this mutoscope and biograph turns so quickly. But if we may judge the consulting physicians by their writings, they are no more agreed as to the best drugs to use than the rest of us. Their articles give us the choice of so many prescriptions—sedative, antiseptic, astringent, alkaline and acid—that it is the story of the Sibylline books reversed; with every moan we give to the continued fatality of this disease, we have another drug suggested which diminishes our estimation of the value of all the former ones.

It is objected that diarrhœa is not a disease, that it is only a symptom. Then we must call it a complaint; it has well earned a place under that heading, for yearly in August our eyes, our ears, our noses, are all appealed to by its importunate complainings.

On many points concerning acute infantile diarrhœa medical men are agreed. All acknowledge its frequency, its fatality, and its annual season in the hot weather. All seem agreed also that a certain percentage of the cases are apparently stricken with death from the first, and that all our efforts are in vain. It is still more certain that the great majority of cases get well

under very various and even almost opposite methods of treatment, so perhaps one may surmise that they would have recovered without any special medicinal treatment, and that they owe their recovery to the expulsive powers of their own muscular stomach and intestines acting upwards and downwards and effecting the removal of all irritating food and intestinal products.

But with regard to many most important points in the treatment of this complaint medical men differ widely. The present writer lives at the seaside and has had considerable experience in these cases, for it seems as if almost all the babies of England are centrifugalised to the edge of the land in August; and yet when he mentioned his present ideas of treatment to a medical acquaintance of more experience and greater authority, the latter expressed his interest in hearing them as his own methods were almost diametrically opposite. If one asks several medical men what drugs they use in these cases, here are various kinds of reply:—

Dr. A.—“I always start with a mixture of catechu, chalk, and a little opium, and I consider that it does good.”

Dr. B.—“I never find astringents such as chalk and catechu useful, and do not think opium is safe. I use small doses of liq. hydrarg. perchlor. or else sod. salicyl.”

Dr. C.—“Bismuth and soda, with a carminative such as sp. chlorof. are the only drugs I use. As diet, I prefer weak animal broths.”

Dr. D. uses a separate drug for each variety of coloured motion. A green motion calls for grey powder, a red motion for ipecacuanha, a pale motion for perchloride of mercury, and a dark motion for something else. This plan seems easy enough, and yet the colour of the remedy compared with that of the motion does not seem to be exactly similar nor exactly complementary. However, it “goes with it.”

With all the yearly experience gained by every practising medical man and every out-patient department, the cases varying but little from a few well known types of the disease, surely one would have thought that at any rate the principles of treatment would have been agreed upon by now. But it is not so.

Mercurial or other aperients, antiseptics, acids, alkalies, enemata, all have their advocates; and the foods selected as suitable under the same conditions by different medical men are very different. Here are opinions to choose from:—

“All milk is bad.”

“Peptonised milk is good.”

“Animal broths, especially veal, are suitable.”

“Animal broths often cause sickness, hastily prepared (as they often are) they contain fat which disagrees.”

“Barley water is at any rate harmless.”

“Barley water is slightly laxative.”

“Whatever you choose as food, let it be simple. Try only one thing at a time, and unless it is evidently causing vomiting, give it at least twenty-four hours trial.”

“Use in turn every two hours the following three foods:—Weak Benger's food, Valentine's meat-juice and water, white of egg beaten up in water.”

“Give extremely little food for two or three days.”

“The child is very weak, and as soon as ever the sickness has stopped, get down as much nourishment as you can consistently with not causing sickness.”

“Give as little fluid by the mouth as possible. It only sets up peristaltic action, runs through the bowels and exhausts the child by frequent motions.”

“Give, unless there is sickness, plenty of water to drink. So far as it is absorbed it replaces the water sucked out of the tissues by the diarrhœa, and what is not absorbed will help to cleanse the bowels.”

“Give for a time weak albuminous food such as white of egg; then carbohydrate food; the bacteria which have flourished on the first diet will die on the second diet, and when a new starch and sugar-loving progeny have appeared and are doing damage, change the diet to an extract of meat, and in turn all the classes of bacteria will be starved out.”

In addition to various medical opinions which solicit his consideration, the young practitioner will be beset by the suggestions as to diet which come from various relatives of the child, possibly

themselves mothers who have lost children by the same complaint, and whose opinions are as weighty as their treatment was unsuccessful. Their diagnosis will probably be "consumption of the bowels," and their treatment arrowroot. It is clearly important for the doctor to have emphatic notions of his own of some sort if he is to retain hold of the reins. Any sign of doubt in his countenance as to which path he should take may result in the reins being taken in charge by stronger hands either domestic or medical.

The medical man will probably be asked as to the cause of this disease. Great authorities say that chills will cause it. It is impossible to prove a universal negative in this case, but that method of causation is hardly suggested by one's yearly experience that it is quite exceptional to be consulted about any infantile diarrhœa in the winter time (when there is plenty of bronchitis), but that in August, out of fifty babies ill forty-nine have diarrhœa or sickness and diarrhœa. Chill has lost caste as a troubler of the peritoneum, the outer and (one would have thought) the more susceptible covering of the bowels, but still appears in repute as a disturber of the mucous membrane.

Heat would seem more likely to be the cause than cold. These cases occur in August, not in January, and apart from the direct effect of heat on the baby, whatever that effect may be, there can be no doubt as to the bad effect which the heat and dust of August may have on the baby's food. From the cow's udder to the baby's mouth milk has a dangerous journey—dirty cow, milk-pail, milk-can, railway movement, street dairy, milkman's can, the milk-jug with dust blown on to it from the open window of the area kitchen, the feeding-bottle with its lengthy and imperfectly cleaned tube, the hot bedroom containing the food for the night or early morning—surely it is not assuming much to guess that in most cases, if not in all, a dose of germ-laden or partly sour milk has started the trouble. Of course, in many cases food evidently improper at the baby's age, even if its quality be good, has been given, such as porridge to a tiny baby or bites of cherry or plum to a one year old. When a baby has diarrhœa and the cause is in question, "*cherchez la nourriture.*"

In August, at the seaside, mothers and nurses fancy that babies can digest anything (especially if the feet be dipped into the sea). Mashed potatoes with a little Worcester sauce, little tastes of shrimps have been given with bad result. "He seemed to like it," is considered sufficient justification for all such experiments.

The writer has not the slightest doubt that the wisest course to adopt in the treatment of all cases is to assume that something taken in at the mouth has caused the trouble, and therefore the first step is to give an aperient to remove all irritating matter.

A medical critic may say that this only need be done when such symptoms as griping pains (shown by screams and drawing up the knees) and ineffectual strainings prove the presence of irritating matter.

In reply to this it can be urged that the two symptoms just mentioned are not the only ones that suggest the presence of an irritant. Does not the diarrhœa itself show continuance of irritation of some sort, and even if the original offending article has been removed (of which it is most difficult to be sure) has not food of some kind been given since, and may not this be continuing the irritation? To warrant the use of an aperient it is not necessary that there should be present in the bowel such things as a piece of pear or a decomposing shrimp; when once the bowels are upset in their process of normal digestion, even good food, such as sterilised milk, will be only partly digested, and will then undergo some process of abnormal decomposition and will become the nidus of bacterial swarms which will produce poisonous matters of one sort or another, and the sooner one can remove by an aperient the effete food and the bacteria and their poisonous productions the better. The bowel must be emptied and then rested, and rest can only properly be obtained by maintaining the bowel in a state of emptiness.

When first called to see the baby at the commencement of the illness or during its course, it is not necessary to waste one's energy by entering much with the mother into the question of past errors in feeding. Whatever the diet has been in the past the child is now ill and the diet must be completely altered for the present; it is no longer a question of what constitutes

suitable food for a baby of a certain age, but of what food can be assimilated by the digestive apparatus in its present irritated and inefficient state. If the baby is going to die, our careful disquisition on the feeding of infants will not be needed; and if, on the other hand, we manage to get the child well, our criticisms on the past and our instructions for the future will be listened to more attentively and perhaps obeyed; and in the details of present treatment (suggested below) such matters of dispute as feeding-bottles and their tubes, and milk from one cow or from forty, fresh or condensed, good, bad, or indifferent, will be temporarily lost sight of.

Our main equipment as a start need not consist of more than a teaspoon and a cup as implements, brandy and castor oil as medicines, and water previously boiled and white of egg as food, with perhaps a rectal and a hypodermic syringe.

Of course the stools must be inspected.

As the purpose of this paper is treatment of disease rather than its description, we need not enter much into the distinctions between the varieties of diarrhœa.

Three chief kinds are noticeable :

1. Simple (mild).
2. Inflammatory (stools often bloody).
3. Choleraic (septic).

But the simple may become inflammatory or choleraic; the inflammatory and the choleraic may be combined; the inflammatory may become choleraic, or if the shock of a choleraic attack be survived, an inflammatory attack may ensue.

However, so far as the diagnosis of simple, inflammatory, or choleraic diarrhœa is to bear on our treatment, the following points may be noted :—

1. If we think we have a simple mild attack to deal with, we had better treat it as carefully as if worse symptoms were to follow on in a day or two.

2. If we think the attack is inflammatory, the temperature (preferably in the rectum) should be carefully watched so that it may be kept within bounds by tepid sponging, and it is probably

in the cases which present bloody motions that ipecacuanha has done good.

3. If from the sudden sunken state of the baby we consider the attack to be one of choleraic diarrhœa, then the condition and treatment of the baby is for the time of more importance than the condition and treatment of the baby's bowels; shock, collapse, prostration, is the danger. The nervous system is poisoned and struck down by shock, the vascular system is partly emptied, and life may be a question of hours. The baby loses weight like a wet sponge when squeezed. Into the treatment of this condition of collapse it is not the writer's purpose to enter fully, for there is hardly any divergence of opinion about it; some practitioners are more energetic in their efforts than others, but the ideas are the same and find vent in the following list of agents:—Stimulants, such as brandy up to ʒi. in twenty-four hours, by mouth, by rectum, and hypodermically; hot baths with or without a tablespoonful of mustard; cotton-wool pads and flannel clothing; hot water bottles near the feet and in the bed; rectal injections of warm saline solution, as much at a time and as often as can be retained, *e.g.* ʒiv. at a time of common salt solution (ʒi. — Oj.); strychnine injections hypodermically $\text{gr. } \frac{1}{400}$ every hour; morphia injections hypodermically $\text{gr. } \frac{1}{100}$, combined with atropine $\text{gr. } \frac{1}{800}$ every hour. The latter (morphia) is said by some authorities to be the best remedy for collapse; it may be so. As to the safety and value of the strychnine and morphia in this condition the present writer is not competent to judge, but he is competent to advise a young practitioner who is inclined to inject these strong remedies into tiny babies balanced between life and death only to do so after dividing the responsibility with another medical man. If the child is not already in a state of stupor, it soon may be, and the hypodermic injection may be blamed, and its donor.

Bearing in mind, therefore, that collapse whenever seen must be energetically treated at once, we begin the treatment of the bowels by an aperient selected say from the following list:—Castor Oil ʒi. , Tinct. Rhei ʒxv. , Calomel gr.j. , Hyd. cum Cret. gr. iij.

If the child has been recently vomiting, wait two hours since the last vomit before administering the aperient or anything else at all by the mouth. Even the remedies for collapse must be rectal or hypodermic. The sickness must be stopped for several reasons :—

1. It is most exhausting.
2. Until vomiting has ceased the aperient will not be able to be given, or if given will return.
3. Until vomiting has ceased the stomach will not retain the remedies for collapse, such as brandy.

Washing out of the stomach, or giving an emetic such as *vin. ipecac.* \mathfrak{z} i. followed by warm water may be a good preliminary in some cases, but these measures are depressant and therefore most undesirable unless absolutely necessary, and the stomach is usually empty if there has been vomiting, and all it needs is a rest.

It is during this waiting stage that one's efforts are most likely to be thwarted by domestic helpers. If very energetic they must be kept employed going to the chemist's for something which he has not got, or preparing external remedies such as mustard baths, or they will never leave the child's stomach alone for two or more hours. Assure the mother that the baby will not die from want of food ; assure her that if it dies, it dies poisoned and not of starvation ; and if she can be convinced of this and got to act upon it, a great danger is disposed of.

Fasting is the best remedy for vomiting.

Sickness having ceased for two hours, the aperient is now given. Should it be vomited, wait again two hours and repeat a similar or a different aperient dose. Thus, if castor oil was the first and was vomited, calomel may be tried next ; but in the writer's experience, if the fasting has been strict, it is very rare for the castor oil to be rejected a second time. Give nothing whatever by the stomach, neither food nor medicine, till the aperient has been kept down at least two hours. Write all instructions down on paper, put the paper on the mantelpiece, and catechise the mother from the paper at each visit. It is

difficult to insist too strongly on the importance of the early emptying and subsequent rest of the stomach and bowels.

The aperient having temporarily cleansed the bowels, and the vomiting having ceased, the stomach now resting, the question arises in the doctor's mind, what shall be put into it? (The question will already have been asked by the mother several times.) In all probability, the less that is put into the stomach the better. The sickness must not be restarted.

Unless collapse is markedly present continue the fast for several hours. Besides being the best remedy for the sickness, fasting is also the best early remedy for the diarrhœa.

But if collapse is urgent, stimulants are badly needed, and therefore the experiment must be tried whether the stomach can retain such things as either teaspoonfuls of warm weak brandy and water or champagne and water, but not food of any kind.

The less feeding there is for forty-eight hours the better. If the medical man is convinced that this is the best course to take he may still have difficulty in convincing the friends of the baby. The bowels are irritated, probably coated with mucus and catarrhal products, possibly they are inflamed and oozing blood, how much food can they digest, and how much of it will decompose and form a pasty, foul-smelling, poisonous, irritating mass? If much feeding be persisted in, it will probably cause vomiting, which after all is perhaps safer than its retention, but more wasteful of the child's strength than fasting would be. The child will cry from fasting, but it will cry from pain if it be fed, and it had better cry and get better than cry and get worse. If food be unfortunately given and kept down, it is likely to cause pain from rapid and irregular peristaltic action, and worse still, it will decompose into a germ-nursery which manufactures poisons causing the various symptoms of collapse, fever, fits, stupor, and the rest.

The theory of poisoning is well supported by the fact that some of the worst cases may not have nearly so many bowel-actions in the twenty-four hours as some cases that recover; and some cases, even after the sickness and diarrhœa have diminished or

ceased, become comatose and die as if they were in the last hours of tubercular meningitis.

The child will not get much benefit from the food, but the bacteria will. The child may get well whilst food is being given, but it is more likely to be in spite of the food than by means of it. Starvation is not the great danger, nor food the great remedy.

By this time we will suppose that the bowels have been emptied, the vomiting has ceased, the collapse is being energetically treated, and that it is thought time to give a little weak nourishment. White of egg thoroughly well beaten up and then diluted with plenty of nearly cold water is as suitable and more easily to be obtained than anything else. Whey is another excellent food. But for simplicity and readiness the white of egg solution is unrivalled. The writer had under care a baby of twelve months old who lived almost exclusively on it for eight weeks. At one time the baby was so ill that the mother and two of her relatives thought it was actually dead, and had covered its face over with a handkerchief. For eight weeks every attempt at adding to the diet caused return of sickness or diarrhœa or both. (The last sentences sound as if they had been cut out from a pamphlet about somebody's meat-juice, but it is not so.) Towards the close of the period the child took from seven to ten whites of eggs in the twenty-four hours, two eggs in half a pint of water. During the early days of acute infantile diarrhœa, begin with a teaspoonful at a time every half hour or hour, and gradually increase the quantity given at a time.

Unless there is vomiting, the supply of water by the mouth need not be limited. If absorbed, it will tend to remedy the collapse by refilling the child's blood-vessels; if not absorbed, it will assist in washing out the bowel.

This matter of keeping the bowels as clear as possible from food residue is of the greatest importance throughout the whole attack. The more pasty and foul the motions are, the less food should one give, and the more frequently should aperients such as castor oil be given so as to ensure early removal of the waste. Many medical men agree with the use of a preliminary purge, but do not seem to see the need for its repetition. The castor oil

is mechanically the most soothing aperient for this purpose, and may be given every day or every other day, a teaspoonful at a time, or two or three times a day a few drops at a time. Small babies seem rather to like it.

The white of egg is also a very soothing application. One can hardly conceive of two things less irritating to a sore bowel than castor oil and white of egg solution, and in the writer's opinion they are for their simplicity, readiness, and efficiency worth, together with the addition when necessary of brandy, in these circumstances more than all other foods and drugs that he has tried put together.

Under this process of having the in-take strictly limited, and of having the waste removed early by castor oil, if necessary, daily, the child wastes as in other methods of treatment, and the crying is not much less, but it is the whining of hunger rather than the cry of pain; and the writer has thought that the tongue remains cleaner and moister, and that the temperature does not seem to range so high as in other methods of treatment, and there is hardly ever the trouble of vomiting. It certainly seems to him the likeliest way of getting the child over the first week of poisoning.

Breast-fed babies (although subjected to the same chills and heats as the others) do not get this complaint so often nor have it so severely as the artificially fed. The inference is obvious. The writer has not had in his own practice a fatal case of diarrhœa in any baby whose sole food was the breast. If a breast-fed baby gets diarrhœa it is usually due to some tit-bit. In any case, abstinence from the milk is advisable for two to three days in order to be on the safe side. The mother can preserve her future power of supply by the temporary use of a breast-pump.

All cases of infantile diarrhœa must be seen frequently, and the buttocks inspected daily. The baby should never be awaked for food or physic; it may happen that the changing of the napkin may awake it, if so, it cannot be helped, that must be done.

Next, and this is most important, keep the child as much as possible all day long in the open air, for it is probably August. It will cry less and sleep more, and even if it has to die may just as well die carefully wrapped up in a perambulator, with hot water bottles near it, under the shade of a tree in the garden or on the balcony at the seaside, as in a stuffy room smelling perhaps of vomit or pasty motions: but it must be a private garden, not a public one, or the sympathetic suggestions as to diet given by other mothers or nurses if the child is open to their inspection will drive the mother frantic and harass the doctor at his next visit.

Whether the practitioner considers diarrhœa to be infectious or not, it will be well for him to make sure that any napkins which are saved for him to see are not kept under the bed, nor anywhere in the child's room, and that they are soon boiled.

The writer has not tried irrigation of the colon with one to two pints of saline solution, but very strongly approves of the idea on which it is based, and which he has tried to emphasize, namely, that the cleansing and keeping clean of the bowels should be effectually done. The rectal saline injections, a few ounces at a time for absorption, mentioned previously as one remedy for collapse, is of course a different matter. The writer has used these with benefit. Injections for either rectum or colon may be used cold if the baby is too hot, or fairly hot if the baby is too cold, or just warm if the baby is neither too hot nor too cold.

The question of the return to ordinary diet after the attack will not now be discussed; it is important, but the mother often relieves us of all responsibility in that respect by giving milk or other baby food long before we should be disposed to allow it. If such an experiment succeeds, the medical attendant will be informed of it and expected to praise her enterprise; but if a relapse results, he will probably be left to flounder about in search for the cause.

We now come to the question of non-aperient drugs. If it were not that some such drugs are invariably given, the writer

would say that this is the least important part of the whole matter.

In the first week of acute infantile diarrhœa do not give any medicine with the view of directly checking the diarrhœa. If it be allowed that castor oil or something similar may have to be given from time to time in order to keep the bowels free from the irritation of waste food-products, and to keep the system free from poisons derived from the same source, surely all astringents which are given to tie up the bowels must be bad. If a child has a pea in its wind-pipe we do not give medicines to allay its cough, troublesome as that may be. Then why, if a child has decomposing stuff in its bowels, are we to try to keep it there merely because it causes frequent motions?

Hence, during the first week at any rate regard as bad (not merely harmless, but bad) all astringents such as chalk, kino, catechu, logwood, rhatany. So far as chalk is merely an alkali, it may be useful to the stomach (but that is not the reason of its use), and so far as it is merely a mechanical soother it may be useful for the bowels, but so far as it is a cement to stop them up it is bad. As an alkali, bicarbonate of soda can replace it, and as a mechanical varnish bismuth is as good or better. No drugs should be given with the view of reducing twenty motions a day to ten, but on the contrary, let the motions come; replace the loss of water from the system by giving water by mouth or by rectum, and diminish the number of motions by diminishing the amount of irritating stuff in the bowels. One would think that the stench of the motions would suggest that they should be removed rather than retained. We are glad enough to get them out of the room, why should we advocate their retention inside the baby?

Now about Opium in these cases. A third of the profession says give it, a third says don't give it, and a third sits on the fence and says that opium needs careful administration. Why is this? Because some things that opium does are good and some are bad. So far as opium is a nerve-soother, allaying pain and fretfulness and diminishing the effects of shock and procuring

rest, it is good. So far as opium is an astringent, paralysing the movement of the intestines, it is bad. And there is another peril sometimes possible. In these cases of acute infantile diarrhœa there is, at any rate, occasionally dropsy, probably anæmic from nephritis or renal defectiveness of some sort, opium is surely then risky. And there is commonly a final stupor in fatal cases of diarrhœa towards which one hardly likes to contribute a soporific. If opium has to be given, it is best given in a starch enema; in that way perhaps it will not act so astringently upon the bowel and it will be as soothing to the nervous system. The writer prefers not to use it and seldom finds it needed for pain if the bowels are kept almost empty and the baby is kept warm.

Next with regard to Mercurials. Calomel is useful as an aperient if for any reason (such as vomiting) it be preferred to castor oil. A preliminary clearing dose of a grain may be given, and subsequently gr. $\frac{1}{4}$ every few hours for two or three days. Or hyd. cum cret. may similarly be given in a preliminary dose of gr. iij., and subsequently gr. $\frac{1}{4}$ every few hours for two or three days. But these powders have such a way of jumping on to the carpet when the paper is opened. Are they better than castor oil? They are less ready. See how moist the lips and tongue are after castor oil.

Bismuth, on the whole, is the favourite drug. It seems at any rate to be harmless; it is antiseptic; its astringency cannot be great, and it is a mechanical varnish; ʒij. daily should be given.

Bicarbonate of Soda may be useful for the stomach; but with the acid and alkali question the writer is not competent to deal. He has never tried acids during the first week of infantile diarrhœa. What are we to do when one authority tells us that the familiar green motions are due to a specific bacillus and that acids (such as lactic acid) should be given to kill it; whereas another authority assures us that the popular alkalies (such as soda) are useful for neutralising the irritating organic acids (such as lactic acid) produced by fermentation in the bowels, and that it is these acids which cause diarrhœa by irritating the bowels?

Tradition at Guy's says that at the close of several *Materia Medica* lectures concerning the actions of acids and alkalies given by one of our physicians, one of our most intelligent students went down to the lecturer's table and quietly asked, "But, Dr. M., when *would* you give acids and when *would* you give alkalies in stomach troubles?" Dr. M's. reply was, "Mr. W., if you have really followed my lectures as closely as you appear to have done, you will have perceived that I am quite unable to tell you."

Of carminatives, brandy is the best.

And now, castor oil or the like having been advocated, acids and alkalies having been mentioned, bismuth having been approved of, and all astringents (including opium) having been disapproved of, it remains to speak of antiseptics such as corrosive sublimate, salol, salicylates, thymol, resorcin, naphthol and many others recommended for their germ-killing power. The writer has tried some of them, but has not been able to form definite conclusions as to their value. The same drug is praised by one man and considered as valueless by another. The great value of any one of them is not widely accepted. Difficult although the task may be, yet will it not be easier and safer to remove mechanically *en masse* the bacteria with the decomposing food and mucus in which they swarm by some such methods as those advocated above than to kill the bacteria by antiseptics? The baby is only a big bacillus and may be killed. How much antiseptic lotion would be necessary to disinfect merely the surface of several feet of tortuous slime-lined gut to say nothing of disinfecting the intestinal contents? However, we may hope that some day there may be found some germ-killing and baby-saving drug which shall prove more universally satisfactory than any we at present possess.

SUMMARY.

Diarrhœa is due to something in the diet.

Empty the bowels and keep them cleansed, preferably by castor oil.

Energetically treat the collapse, and don't trouble about the number of motions.

Don't stop up the bowels by astringents and opiates.

Give hardly any food for a few days. Begin with white of egg solution in teaspoonful doses every half hour.

If the baby is too hot, cool it ; if it is too cold, warm it ; if it is thirsty and not sick, give it water ; and keep it in the fresh air all day long.

AN INVESTIGATION OF THE CHEMICAL COMPOSITION OF A SPECIMEN OF COLLOID CARCINOMA OF THE OVARY.

By L. C. PANTING, M.D.

THE specimen examined was a very large colloid carcinoma of the ovary obtained at the autopsy of a patient under the care of Dr. Pye-Smith.

The colloid material was found in loculi separated from one another by a thin transparent membrane, and could readily be shelled out as a light yellow jelly on cutting through the wall. The jelly itself was transparent, but contained threads of a tough, white substance, which appeared to be fibrous tissue. The material in different loculi was not quite identical, some portions having a much deeper colour than others: the least coloured portions were as far as possible selected, and could be obtained quite free from contamination with blood, as very few vessels were present in the wall of the cyst.

When dried at 100° C. until it no longer lost weight, a yellowish white powder was formed, which rapidly absorbed moisture from the air and gradually swelled up in water to a jelly resembling the original colloid substance.

It was found to contain water, 90·86 per cent. ; organic matter 8·07 per cent. ; ash, 1·07 per cent.

The ash contained sulphur and phosphorus (and, of course, carbon dioxide) in combination with sodium and calcium.

The phosphorus amounted to 0.2 per cent. of the dry solid, and, as none was found in the compounds afterward obtained from the colloid, it was probably present only as inorganic phosphate.

Since iodine has been found in substances derived from the thyroid gland, it seemed possible that it might be present in colloid from other sources also. A considerable amount of the dry colloid was accordingly fused with excess of caustic alkali, and the resulting mass examined in the usual way, but no trace of this element could be discovered.

The original colloid was in part soluble in water, whilst the residue dissolved in dilute alkali. The alkaline solution so obtained possessed the power of reducing Fehling's solution when simply boiled with it, but this power was greatly increased if the liquid had previously been treated with acids.

Thus the investigation resolved itself into an examination of the part soluble in water, and of the part soluble only in dilute alkalis, and possessing reducing properties; and finally into the isolation as far as possible of the reducing substance itself.

It has long been known that colloid of the ovary and cysts of that organ contain substances possessed of reducing properties. One of these was described by Scherer (*Wurtzburger Zeitsch.*), in 1866, and more fully investigated by Hammarsten (*Zeitsch. für Physiol. Chemie.*), in 1882, who gives the following description of its properties:—

It is soluble in water, forming a somewhat mucoid solution, which does not coagulate on boiling, nor form a precipitate on the addition of acetic or hydrochloric acid; with nitric acid the solution assumes a milky appearance, but on the addition of more acid it clears to a yellow liquid. When potassium ferrocyanide is added to the acetic acid solution, the liquid becomes opaque, but no definite precipitate is formed; Millon's reagent causes a reddish brown colouration; with glacial acetic and sulphuric acids the liquid assumes a violet tint; saturation with magnesium sulphate does not throw it out of solution even on boiling. On the addition of four volumes of alcohol the substance is completely

separated as a white, thready precipitate, which retains its solubility in water after standing a month under alcohol. It has no direct reducing action on Fehling's solution, but after it has been boiled with dilute acids the solution reduces alkaline copper. It contains 10·26 per cent. of nitrogen and 1·25 per cent. of sulphur. To this substance, which had been known as metalbumen, Hammarsten assigned the name pseudomucin.

In 1895, a compound similar in many respects to pseudomucin, but differing in that it reduced Fehling's solution directly, was described by Mitjukoff (*Archives für Gynäkologie*, 1895), who obtained it from colloid of the ovary that had been washed in water and dilute hydrochloric acid. It dissolves partially after prolonged boiling with water, but appears to be decomposed in the process. It is insoluble in cold water and dilute acids, and in sodium carbonate solution, but it dissolves slowly in dilute caustic alkali, forming a solution which is at first colourless but soon becomes yellow. It contains 10·5 per cent. of nitrogen and 1·13 per cent. of sulphur. The solution in alkali yields a precipitate on neutralization with sulphuric or acetic acids which gives all the proteid reactions—xanthoproteic, biuret, Millon's, and Adamkiewicz's. This precipitate contains 13·7 per cent. of nitrogen, and is looked upon by the author as an albuminate. The filtrate yields a precipitate with alcohol, which re-dissolves in dilute alkali, and this solution gives all the reactions of the original alkaline solution, with the exception that the precipitate with acids is only formed when the liquid has been allowed to stand before the addition of the acid. When ferrocyanide of potassium is added to the acid liquid, a precipitate slowly develops; hydrochloric acid causes a precipitate soluble in excess, whilst that produced by sulphuric acid is not soluble in excess. Nitric acid throws down a precipitate which dissolves in excess to form a brown solution. No precipitation but only a greater opalescence is caused by boiling in alkaline neutral or acid solution. Fehling's solution is directly reduced, but more marked reduction is obtained after previous treatment with dilute acids or alkalis. The solution gives the biuret and xanthoproteic reactions, a purple colour with glacial acetic and sulphuric acids and a red

colour with Millon's reagent. To this substance the name paramucin has been given.

The same author (*loc. cit.*) has also obtained a similar compound from the strongly alkaline liquid of an ovarian cyst, which, in addition, contained alkali albumen; this substance, however, after precipitation by alcohol, remains soluble in water. It gives the usual reactions of pseudomucin but reduces Fehling's solution directly.

The paralbumen which was described by Scherer has been shown by Hammarsten to be a mixture of pseudomucin and albumen.

Such are the substances which occur in ovarian colloids and cysts, and appear to be peculiar to them. They are clearly associated with the mucin group in their general reactions, and in particular in their power of reducing alkaline copper either directly or after previous treatment with acids.

The substances so described do not, however, appear to be quite identical in the specimens examined, as, for instance, the two forms of paramucin just described, one of which is soluble, whilst the other is insoluble in water. Still they at least represent types, with which the substances found in the specimen of colloid forming the subject of this paper must be compared.

These substances will now be more fully described.

The original colloid was broken up as far as possible and allowed to stand in water for twenty-four hours, when it was found to have swelled and partially dissolved. The gelatinous portion was separated by filtration through muslin, and the liquid could then be filtered in the ordinary way.

The substance obtained from the filtrate will be called A. The residue was washed with dilute hydrochloric acid, and then with water until the wash water no longer gave an acid reaction. So obtained it formed a bulky almost diffuent mass, which was insoluble in water even after prolonged boiling, remaining apparently unaffected by the process, and showing no signs of coagulation.

It was insoluble in dilute acids, and in weak saline solutions; it was unaffected by ether, and became shrivelled when treated

with alcohol; it dissolved slowly but almost completely in 1.5 per cent. solution of sodium hydrate. The liquid was filtered from the minute residue of insoluble substance, which appeared to consist chiefly of the fibrous threads noticed in the original colloid. The solution was clear and colourless and remained so for several weeks. It was precipitated by alcohol, and this precipitate will be called substance B.

Substance A.—The solution of substance A, obtained directly from the colloid, was colourless and transparent, and only slightly gelatinous; it could be filtered without difficulty. It was faintly alkaline to litmus, and on neutralization with acetic acid became milky, but no deposit could be seen after allowing the liquid to stand for several days, nor was the opalescence diminished by filtration; it became less marked, however, when more acetic acid was added. A similar opalescence was produced with sulphuric acid, but again no definite precipitation. No coagulation occurred on boiling the liquid either in the alkaline, neutral, or acid state, but a film formed on the surface when an attempt was made to concentrate it. The solution gave the xanthoproteic reaction; with glacial acetic and sulphuric acids a fine purple, and with Millon's reagent a deep red colour was produced. The solution, rendered opalescent with acetic acid, was precipitated by the addition of potassium ferrocyanide. With nitric acid a white precipitate was produced, and with copper sulphate a bluish-white precipitate, which was soluble in excess of caustic potash to a purple solution.

The solution did not reduce Fehling's solution, either directly, or after preliminary boiling with dilute sulphuric acid; no precipitate was formed on saturation with magnesium sulphate, but on saturation with ammonium sulphate the substance was precipitated completely.

The precipitate so obtained, when dissolved in water, formed a solution which coagulated on boiling, the coagulation point being between 75° C. and 77° C. With five or six volumes of alcohol complete precipitation also took place, the precipitate dissolving in dilute alkali to give a solution identical with that from which

it was obtained. After standing under alcohol for twenty-four hours the precipitate was found to be insoluble in water.

These precipitates were examined for phosphorus in the usual way by fusion with caustic alkali, but none was present.

Colloids being in the main insoluble, the soluble substances occurring in them, do not seem to have received much attention, presumably because the assumption has been made that they consist only of lymph diffused through the substance.

The substance A, however, is not precipitated on neutralization, nor is any coagulum formed on boiling its solution. If, then, as appears probable, we are to look upon it as an albumen, we must assume that some substance is present in the original liquid which prevents coagulation. This substance must be soluble in a saturated solution of ammonium sulphate, since the albuminous precipitate so obtained, when re-dissolved in water, coagulates readily. Of the substances known to be present, the alkaline phosphates are the only ones recognised as in any way possessing this property, and their presence seems hardly sufficient to explain the whole reaction when excess of acid is present. The complete absence of globulin from the solution renders its origin from lymph exceedingly improbable, and moreover it seems far more likely that both the albumen and the colloid substance are derived from the decomposition of some more complex compound. This seems the more probable since many specimens of colloid have been shown to break up readily into an albumen and a compound of the mucin type, to which this colloid clearly belongs.

Substance B.—Some of the washed colloid was dried at 100° C. until it no longer lost weight. It then formed a white powder which rapidly absorbed moisture from the air, and when treated with water swelled up gradually to an insoluble jelly resembling the original colloid. It dissolved slowly in alkalies, and this solution had the same reactions as that obtained directly from the colloid.

The nitrogen content was determined by Kjeldahl's method to be 14.95 per cent.; sulphur and phosphorus were sought for by fusion with potash in the usual way, but none was present.

A solution of the washed colloid in 1·5 per cent. sodium hydrate was found to give the following reactions :—

On neutralization with acetic acid no precipitation occurred, but the liquid rapidly became cloudy, the cloudiness being greatest when there was a distinct excess of acid. A large excess, however, diminished the opalescence, although the liquid never became quite clear. With dilute sulphuric acid a similar result was obtained, but boiling caused the liquid to turn brown and a flocculent precipitate to form. With nitric acid the typical white ring of albumen was formed at the junction of the liquids, but, on warming, this precipitate dissolved, and the solution gave the xanthoproteic reaction with ammonia. With acetic acid and potassium ferrocyanide no precipitation occurred: with Millon's reagent only a yellowish colouration appeared; with Fehling's solution some reduction took place after boiling for a few minutes, quite definite, but only small in amount. After treatment of the solution with dilute sulphuric acid at 100° C. for half an hour, the reduction with Fehling's solution was very much more marked. No precipitation was caused by saturation with magnesium sulphate, but on similar treatment with ammonium sulphate, a white granular precipitate was obtained which was easily filtered off. The precipitate, re-dissolved in water, gave all the reactions of the original solution, and the filtrate caused no reduction with Fehling's solution.

It would thus seem that no decomposition was caused by solution in 1·5 per cent. soda solution, such as Mitjukoff found in the investigation of a similar colloid.

With alcohol a thready precipitate was produced, but a large excess of this reagent was required for complete separation, This precipitate formed a glutinous mass on the paper, which interfered so much with filtration that it was necessary to allow subsidence to take place for more than twenty-four hours before attempting to start it. After thus standing under alcohol the precipitate only slowly and partially dissolved in water, although immediately after precipitation it was readily soluble; the aqueous solution possessed the direct reducing power and other

properties of the original liquid. The glutinous substance dissolved quite readily if a few drops of caustic soda solution had been added to the water.

The filtrate from the precipitate, when large excess of alcohol had been used, no longer possessed reducing power, nor did it give the proteid reactions, and it may therefore be concluded that the substance B is not soluble in moderately strong alcohol. This point is of considerable importance, as separating it from one of the products of its decomposition, which is possessed of reducing power, but is readily soluble in strong alcohol and soluble even in absolute alcohol. An estimation of the nitrogen in the alcoholic precipitate, made by Kjeldahl's process, gave 15.17 per cent., which is in accordance with the amount found in the washed colloid. No phosphorus or sulphur was present in this precipitate.

The most important difference between the substance B and paramucin, as described by Mitjukoff, appears to be in the percentage of nitrogen in these compounds. In substance B this value approaches nearly to that found in albumen, whilst that in paramucin corresponds to the percentage found in mucin and pseudomucin (10.28). The nitrogen determinations agreed with one another well, so that it seems unlikely that any serious error could have been made. In view of the high nitrogen percentage, it might be suggested that substance B is really only a mixture of a little paramucin with a large amount of albumen. This seems hardly to meet the facts, however, for no neutralization precipitate, or coagulum on boiling, could be obtained, either from the original solution or from solutions of the alcoholic or ammonium sulphate precipitates. Again, if either albumen or albumose were present in appreciable quantity, we should expect to obtain all the proteid reactions; and these could not be obtained from the substance B.

In other respects the resemblance between the substance B and paramucin is very marked. They together differ from all other compounds which have been found in ovarian cysts or colloids, in that they reduce Fehling's solution without preliminary treatment with acids. This reduction must be caused by the substance B

and not by any decomposition product formed at its expense by the action of the alkali originally present in the colloid, for the solution loses its reducing power after precipitation by alcohol or ammonium sulphate. These precipitates, moreover, when re-dissolved show the same reducing power as the original solution, while the reducing substance, obtained from decomposition of the colloid by the action of alkalis, is not precipitated by either alcohol or ammonium sulphate.

To return to the comparison, both paramucin and the substance B are rendered insoluble by the prolonged action of alcohol, and here again differ from pseudomucin, with which they have many characteristics in common. Their relationship to pseudomucin is shown by the fact, that whilst giving many of the proteid reactions none of these substances yield a coagulum on boiling, and that while splitting up on treatment with acids to give a reducing substance, they are not precipitated by acetic acid.

Decomposition of substance B.—In order to split up the molecule, and as far as possible isolate the reducing substance, a considerable quantity of colloid was dissolved in 10 per cent. solution of sodium hydrate, and allowed to remain at the temperature of the laboratory for a fortnight. After dilution and filtration from the insoluble residue the liquid, which was slightly opalescent, and of a faint brown colour, was neutralized with dilute sulphuric acid and boiled. In this process a precipitate was obtained which gave all the reactions of alkali albumen.

After the removal of this precipitate, excess of sulphuric acid was added to the liquid, and it was evaporated on the water-bath until the brown colour began to deepen perceptibly; it was then precipitated with nine or ten volumes of alcohol. The precipitate was filtered off and re-dissolved in dilute alkali, and the solution was neutralized with sulphuric acid in order to separate any alkali albumen which had escaped precipitation. A slight precipitate which formed having been filtered off, the liquid was again precipitated by alcohol, and the process repeated until no further neutralization precipitate could be obtained. The substance thus freed from alkali albumen gave the following reactions:—With

glacial acetic and sulphuric acids a purple colour was produced; with nitric acid, a white precipitate soluble in excess; with acetic acid and potassium ferrocyanide, a white precipitate was produced; the solution gave the xanthoproteic and biuret reaction, but no colouration with Millon's reagent; it was not coagulated on boiling in neutral solution, but was precipitated by saturation with ammonium sulphate; it did not reduce Fehling's solution either directly, or after previous treatment with dilute acid. We may conclude, then, that this substance belongs to the group of albumoses.

The alcoholic filtrate, from which this substance had been separated, was found to possess considerable reducing power, and an attempt was therefore made to isolate the reducing compound in as pure a state as possible.

The greater part of the alcohol was distilled off on the water-bath, but as, after a time the liquid began to rapidly darken in colour and acquire the odour of burnt sugar, this method of concentration was given up. Distillation in vacuo was then tried, but proved impossible owing to the frothing of the liquid.

As the liquid had by this time been reduced to a moderate bulk, it was neutralized with barium carbonate, and after filtering off the barium sulphate and excess of carbonate, the liquid was evaporated in vacuo over sulphuric acid at a temperature of 30° C. The thick brown syrup, obtained in this way, was mixed with crystals of sodium sulphate, although the greater portion of this substance had separated out on the addition of alcohol to the solution. This magma was treated with strong alcohol, which dissolved the brown syrup, but left the crystals behind, apparently free from any precipitated proteid material. The alcoholic solution was again evaporated at the ordinary temperature, but even after leaving the syrup in vacuo for a week it did not become solid. In the hope that the substance could be obtained in crystalline form, the syrup was again dissolved in strong alcohol and an equal volume of ether added. The liquid at once became cloudy, and after a time a thick syrup collected at the bottom of the vessel, and minute crystals formed on the sides. Unfortunately, the crystals were so minute that they could not be

collected ; but they were shown to be insoluble in ether, and soluble in alcohol and water to solutions having reducing properties.

The syrup dissolved in alcohol of any strength but not readily in absolute alcohol ; it was very hygroscopic and mixed with water in any proportion, and it had a brown colour and an odour of burnt sugar. An aqueous solution gave the following reactions :—No precipitate or ring was formed with nitric acid, but on mixing the liquids a violent reaction took place, with evolution of the oxides of nitrogen, the liquid assuming a brown colour ; no reaction appeared to take place with hydrochloric or sulphuric acids in dilute solution ; no characteristic reaction took place with Millon's solution, or any other of the proteid tests. Copper sulphate produced a green precipitate which dissolved in caustic alkali to form a dark purple solution, which even on gentle warming precipitated the copper as cuprous oxide ; ferric chloride produced a deep brown colour ; no precipitation occurred on saturation with ammonium sulphate.

As this substance could not be isolated in the solid state, an attempt was made to convert it into an osazone. The aqueous solution was heated on the water bath for an hour with phenyl hydrazine in acetic acid solution, but no crystalline substance could be obtained. In one experiment a precipitate formed, but microscopically it was found to consist of brown globules, which had no resemblance to the crystals of an osazone. The globules were soluble in alcohol, but no crystals could be obtained from the solution.

The presence of nitrogen in the compound was proved by heating a portion of the syrup with sodium, when sodium cyanide was found in the resulting mass. Now, it is possible that this nitrogen may have any of at least three sources. In the first place, it may be that one of the albuminous substances, into which B has been shown to split up, escaped precipitation, and that this is the origin of the nitrogen. Again, it may have been derived from some of the more extreme decomposition products of substance B, such as the amido-acids, which are known to be formed from proteid at high temperatures by the action of alkali.

Lastly, it may have been derived from the reducing substance C itself.

The first explanation seems improbable, as the syrup was twice submitted to solution in strong alcohol, a process which might be expected to eliminate albuminous compounds, since substance B and all known proteids are insoluble in this reagent. Again, the solution of the syrup in water gave none of the proteid reactions, while a relatively considerable amount of cyanide was formed, which would indicate that more than traces of a nitrogenous substance must be present.

The second possibility, namely, that amido-acids may be present cannot be absolutely denied, but the treatment to which the colloid was subjected does not seem to have been sufficiently severe to produce this decomposition. From these considerations it appears most probable that the reducing substance itself contained nitrogen in its molecule, and that it is allied rather to glucosamine than to glucose. It became of interest, therefore, to determine whether the nitrogen could be eliminated, and the amido-group, if present, replaced by the hydroxyl-group through the action of nitrous acid. Accordingly a solution of the syrup was treated with sodium nitrite and acidified, but no nitrogen was evolved on warming, and no osazone could be obtained from the resulting liquid. From this it would appear probable that the nitrogen is in some more stable position than that which it holds in a simple amine.

Until recently the reducing substances obtained from the decomposition of colloid growths of the ovary have been but little investigated. By earlier observers, among whom may be mentioned Eichwald (*Wurtzburger Med. Zeitsch*, 1864), these substances were looked upon as identical with grape sugar, until Hammarsten showed that they did not ferment with yeast. Mitjukoff (*loc. cit.*), made a careful investigation of the reducing substance obtained from paramucin by the action of acids, but was unable to isolate it. This was due partly to the readiness with which it underwent decomposition with the formation of humus-like bodies—a tendency, it may be remarked, which the substance C also showed—but chiefly to the impossibility of

converting it into crystalline compounds. Although on one occasion a crystalline substance resembling an osazone, and melting at 202° C. was found, yet on repeating the experiment none could be obtained. Mitjukoff's solution was not affected by alcohol, or any other proteid precipitant, and although it gave a faint Biuret reaction, this may be neglected as it was probably due to an impurity. It did not ferment with yeast, nor did it rotate the plane of polarized light.

Leathes (Private communication by the Author from Reports to British Association, Scientific Grants Committee, 1897 and 1898), has isolated from ovarian mucoids a substance, which he believes to be that part of the mucoid molecule which gives to the latter its power of reducing on treatment with acids. This substance contains 5 per cent. of nitrogen, but gives no proteid reactions. It yields on boiling with acids a strongly reducing body (evidently a hexose or a hexose-derivative), an organic base, and formic and acetic acids. The reducing substance obtained by the action of acids forms well-defined crystalline compounds with phenylhydrazine, and these compounds yield on analysis figures corresponding to a simple hexosazone. In some instances he has shown the presence of a paired sulphuric acid in mucoid.

Previous to Leathes' investigation nitrogen had not been detected in the reducing substance derived from paramucin, although it had been found in some of the reducing substances obtained from compounds allied to it, such as mucin and pseudomucin. Thus, Fr. Muller (*Sitzungsber. d. Gesellsch. zu Beforderung d. Gesamten Naturwissenschaft zu Strasburg*, 1896), obtained a substance of this nature by the action of sulphuric acid on mucin occurring in sputum. The mucin was boiled for twenty-four hours with a three per cent. solution of the acid, and in this way he obtained a brownish mass which contained 3.26 per cent. of nitrogen. It was very hygroscopic, reduced alkaline copper solutions even in the cold, was not fermentable by yeast, but had a slight lævo-rotatory action on polarized light. A similar compound also containing nitrogen was obtained by the action of alkalis on pseudomucin.

Muller looks upon these substances as similar in composition to glucosamine.

Hammarsten also (Phys. Chem.) has obtained a nitrogenous reducing substance from submaxillary mucin. In view of the existence of these compounds it is the less surprising to find that the substance B, which clearly belongs to the mucin group, is also capable of yielding a reducing substance containing nitrogen.

The substance B is thus decomposed by alkalies and acids in a manner which would be expected from the similarity of its reactions to those of paramucin. Like this compound it has been shown to break up into an albuminate, an albumose, and a reducing substance. This latter cannot be looked upon as one of the true sugars, but rather as allied to glucosamine, since it contains nitrogen probably in its molecule.

With regard to the reducing power of the substance B, although it acts without previous treatment with acids, this action is slow. The meaning of this may be that the alkali in the Fehling's solution decomposes substance B at 100° C. with separation of substance C, and that this latter then causes reduction of the alkaline copper. This explanation would apply with even greater force to paramucin, as it appears to be less stable in the presence of alkali than substance B. If this view be correct, the relation of paramucin and substance B to the mucin group would be more close than would otherwise appear.

It may be of interest, in conclusion, to compare the general properties of the colloid which forms the subject of this paper, with those of previously described colloids of the ovary and of other organs.

The majority of colloids of the ovary seem to consist chiefly of a substance insoluble in water, but soluble in alkalies, and not coagulated at the boiling point of water. They are all alkaline in reaction, and in some cases the solution in alkali yields a neutralization precipitate, in others it does not, a reaction which appears to depend upon the occurrence or non-occurrence of decomposition. Gautier (Bull. de la Soc. Chem. de Paris, T. XII.), however, examined a specimen which dissolved almost completely in water after prolonged boiling, but it is obvious that this

proceeding may easily have produced some decomposition. A considerable difference in the percentage of nitrogen has been observed in different specimens. Thus, Lebert (Virchow's Archiv. 1852) finds 7 per cent., Gautier (*loc. cit.*) 6 per cent., Mitjukoff (*loc. cit.*) 10·5 per cent., and in the specimen here discussed 14·95 per cent. was found; whilst other observers give values varying from 6 per cent. to 13 or 14 per cent. Sulphur, moreover, has been found in some specimens but is absent in others. The only conclusion which can be drawn from these results is that even ovarian colloid cannot be looked upon as a chemical entity, and this conclusion has been endorsed by all observers.

Among colloids occurring in other organs even greater differences appear to exist, although most of them show the same general reactions as ovarian colloid.

In specimens from the lung, examined by Luschka (Archiv. für Path. Anat. und Physiol., 1852) and Wagner (Archiv. für Heilkunde, 1862), respectively, the colloid had similar properties to that derived from the ovary, and similar results were obtained by Buhl (Illust. Med. Zeitung, 1852) in a specimen occurring in the liver. In another specimen from the liver, however, Virchow (Archiv. für Pathologische Anatomie und Physiologie, 1847) observed markedly different properties, for it had an acid reaction, and was taken up by water to form a partial solution which was not coagulated on boiling, but was precipitated both by acids and by alkalies. The percentages of nitrogen in these colloids also vary considerably, and, again, sulphur is sometimes present and sometimes absent.

There seem, then, grave difficulties in looking upon colloid, as defined by morbid anatomy, in the light of an unit from the point of view of pathological chemistry. It must, however, be admitted that the substances obtained from organs undergoing colloid degeneration are closely allied in nature, whether they occur in colloid proper, or in the fluids from ovarian cysts. The similarity of these two groups was long ago emphasized by Virchow in his suggestion, that these cystic fluids were really formed from colloid by the solvent action of alkali within the body, just as

similar fluids may be formed from it by the action of alkali outside the body.

This view is supported by Pfannenstiel's observation (*Arch. für Gynakol.* 38) that pseudomucin is absent from normal liquor folliculi and hydrops folliculi of the ovary, whereas it is found in glandular and papillary proliferating cysts of this organ, especially when colloid degeneration is taking place. He has also detected this compound in ascitic fluid, usually in association with ruptured ovarian cysts, but occasionally in association with epithelial cancer of some portion of the abdominal contents. Hammarsten (*Zeitsch. für Physiol. Chemie.*, 1891) also has found it in a few ascitic fluids associated with growth of abdominal organs, but in which disease of the ovary could be absolutely excluded, some of the patients being men.

It may be mentioned that I have lately found a trace of a substance giving all the reactions of the substance B in a specimen of ascitic fluid. This fluid was obtained from a male patient now in the hospital under the care of Dr. Hale White, who looks upon the case as probably one of cancer of the liver. It would seem, then, that pseudomucin and allied compounds occur chiefly in association with colloid disease of the ovary, but that colloid disease of other organs, and even carcinoma not undergoing colloid degeneration, may also give rise to it.

It is to be regretted that hitherto the history of the case and even the general morbid appearances have been recorded in but few of the cases, in which chemical investigation has been carried out. In consequence, any attempt to correlate the clinical course of the malady with the chemical characteristics of the growth is out of the question.

The following is the history of the case, which I am enabled to publish through the kindness of Dr. Pye-Smith.

Elizabeth C., æt. 57, was admitted under the care of Dr. Pye-Smith on January 18th, 1898, for swelling of the legs and abdomen.

Pain commenced in the right iliac region four months before admission, and simultaneously a gradual increase in size of the abdomen and swelling of the legs was observed. The swelling

of the legs was only present after standing, and disappeared with rest.

On admission, the abdomen was found enormously distended, with considerable bulging of the flanks, the skin tense, and the umbilicus protruded. The region about and above the umbilicus was resonant, while the rest of the abdomen was dull; the region of resonance shifted to the upper flank when the patient lay on her side. The distension of the abdomen was more marked over the right iliac region, and here a sense of resistance was noticed on palpation. The thoracic organs appeared healthy, and the urine normal, so far as it was examined.

On January 8th, an attempt was made to relieve the distension of the abdomen by paracentesis, but no fluid could be obtained.

On February 4th, it is noted that no change in note occurred on placing the patient on her side, the upper flank remaining dull. From this date on the upper flank seems at times to have remained dull, and at times to have become resonant, on placing the patient on her side.

On February 24th, an exploratory operation was performed by Mr. Jacobson, and a large quantity of gelatinous fluid removed, some gelatinous nodules being observed in the abdomen. The patient recovered from the operation, but gradually got weaker, and died on March 28th. No change in the physical signs was noted from February 24th to the time of the patient's death.

Autopsy.—Lungs: Both bases were collapsed; there were old adhesions and cretaceous material at right apex. Aorta: Patches of atheroma were present. Heart: weight, seven and a half ounces; there were fibrous changes in the substance of the left ventricle fairly well defined at the margin, and some diffuse fibrous change in right ventricle. There were recent vegetations on the aortic valves. Kidneys: weight, eleven ounces. The capsule was slightly thickened and adherent, and a small cyst was found under the capsule of one kidney. Spleen: weight, sixteen ounces; enlarged, adherent to the diaphragm, healthy on section. Stomach, pancreas, and liver were normal.

On opening the abdomen the intestines were seen to be covered with a semi-transparent gelatinous material, and were fixed by general adhesions. Eleven pints of gelatinous fluid were removed from the abdominal cavity. A large gelatinous mass, weighing sixty-seven ounces, was removed from the hypogastric region, which was attached by a pedicle to the right broad ligament. It consisted of a number of globules of jelly with a common centre of attachment.

TUBERCULOSIS OF THE NASAL MUCOUS MEMBRANE.

By FRANCIS J. STEWARD, M.S.

ALTHOUGH this disease is doubtless rare, it is probably not so rare as the small number of published cases would seem to show. During the last three years 2,777 patients have been treated in the Throat and Nose department at Guy's, and in these 2,777 patients there were three cases of tuberculous disease of the nasal mucous membrane.

In his text-book, published in 1884, Sir Morell Mackenzie, wide as his experience undoubtedly was, stated that he had never seen a case of tuberculous disease of the nose, and up till that time only a few isolated cases had been published.

The earliest mention of the disease that I have been able to find was a case recorded by Morgagni in 1765. This was a case of tuberculous disease of the pharynx and palate, in which ulceration of the nasal mucous membrane was also present.

In 1853 Willigk mentioned a case in which he had found tuberculous disease of the septum of the nose, and in 1877 Laveran described two cases in detail. During the following twelve years a number of further cases were reported, and in 1889 Mertens was able to collect in all thirty-one cases in a dissertation on the subject. Since that time many cases have been published by a number of observers, and of these I have been able to collect ninety-four, which together with six cases, now published for the first time, form the basis of this paper. A small number of published cases do not appear in my list either

because the details were so meagre as to render them valueless, or because I have not been able to see the publication in which they were reported.

A number of cases of lupus spreading from the skin to the mucous membrane of the nose, have been published as cases of tuberculous disease of the nose. These cases I have omitted from my list because they are merely instances of direct extension of lupus and in no way differ from lupus occurring elsewhere.

The following are the notes of the six cases, which are now reported for the first time :—

CASE I.—T. D., æt. 36, was admitted under Mr. Symonds in April, 1893, for naso-pharyngeal sarcoma. There was no evidence of tubercle in his family history. The patient had had syphilis twenty years previously, but had suffered from no other ailment till the present one.

Nasal obstruction was first noticed three months before admission, and had gradually increased since that time. On admission, the patient was found to have almost complete nasal obstruction, which was due to a large, soft swelling of a reddish colour which filled the post-nasal space.

On May 2nd the swelling was removed, partly by a snare passed through the nose and partly with forceps. Hæmorrhage was free, and the swelling was soft and easily broke up. It is stated in the report that it was doubtful whether the whole of the growth was removed. The patient remained well for nearly three years, then nasal obstruction again became noticeable, and a lump appeared in the neck.

He was re-admitted in May, 1896, for supposed recurrence. On examination, it was found, however, that the naso-pharynx was quite normal, and that the nasal obstruction was due to enlargement of the inferior turbinated bodies, which was more marked on the right side. There was a glandular swelling below the jaw on the left side. It was of considerable size, and at one point was softened, the skin being adherent.

Portions of both the inferior turbinals were removed, and it was found that the posterior extremity of the right one was enlarged by a distinct new growth, which was ulcerated in several places.

The swelling in the neck was operated on at the same time and some glands were removed and were found to be tuberculous. Microscopical examination of the growth on the right inferior turbinal proved it to be tuberculous.

Since the operation the patient has remained perfectly well. He was seen on January 5th, 1900; the nose and naso-pharynx were then quite free from disease, and there was no enlargement of the cervical lymphatic glands.

CASE II.—K. G., æt. 35, a married woman, came to Throat Out-patients on June 10th, 1898, for nasal obstruction. Father died of pleurisy; apart from this there is nothing of note in the family history. The patient is quite healthy, has had no previous illnesses, and has five children, all of them strong and healthy. Nine years ago the patient had some obstruction of the right nasal duct, and several probes were passed. She does not think she had any nasal symptoms at that time, and after the probing the trouble was cured and she has remained so ever since.

The present trouble began eighteen months ago, when the patient first noticed slight obstruction to the passage of air through the right nostril. Since that time the nose has become gradually more and more blocked, and a slowly increasing swelling has made its appearance. On a few occasions there has been a little bleeding as the result of slight blows, but apart from this there has been no discharge, nor has there been any pain.

On examination, a large swelling is at once seen, completely blocking up both nostrils. It is quite an inch in diameter, and it markedly distends the nose. It is bright red in colour, firm in consistence, and easily bleeds when touched. On the portion projecting into the left nostril is a small ulcerated area about a quarter of an inch in diameter.

The growth springs from the cartilaginous portion of the septum nasi, which is very largely destroyed. The other portions of the nasal cavity could not be examined owing to the large size of the tumour.

No evidence of tuberculous disease elsewhere could be found. The growth was thought to be a gumma, so the patient was ordered Iodide of Potassium in increasing doses till she was

taking twenty grains three times a day. She continued to take this for about two months, during which time the growth decreased slightly. The patient did not attend again till October 28th, when the growth had quite regained its original size. After the application of cocaine a small piece of the growth was removed for microscopical examination. This proved that the tumour was beyond doubt tuberculous. The patient was therefore taken into the hospital by Mr. Symonds and the tumour thoroughly removed by scraping. Sections of the portion of growth removed for examination showed the typical structure and arrangement of tuberculous granulation tissue. Many quite distinct giant cell systems were present, and there was also a large number of epithelioid cells. There was no caseation. The tumour after removal showed the same structure but in parts caseation had taken place.

A large number of sections were carefully stained for tubercle bacilli, but none could be found. In July, 1899, the patient was quite well, and no recurrence had taken place.

CASE III.—A. H., æt. 17, male, was admitted under Mr. Symonds in October, 1895, for stoppage of the right nostril. His family history was unimportant. He first noticed some obstruction in the right nostril eight months before admission. This had gradually increased, and had been attended by some discharge, which was more marked at night and by occasional slight bleeding. On examination, a reddish swelling could be seen blocking the right nostril completely. It was attached to the anterior part of the nasal septum, which it had perforated. The surface of the growth was ulcerated, as was also a portion which presented in the left nostril through the perforation.

There were no signs of tuberculous disease in the lungs or any other part of the body.

The swelling was removed by curetting, and on microscopical examination was found to be tuberculous. No mention is made in the report as to the presence or absence of tubercle bacilli.

The swelling recurred frequently and spread on to the floor of the nasal cavity. It also destroyed practically the whole of the

cartilaginous portion of the septum, including the columna nasi, so that the tip of the nose became depressed.

Later, ulceration occurred on the posterior pharyngeal wall; the lungs and other organs, however, remained quite healthy. The ulceration was treated by repeating curetting and the application of lactic acid. The patient was re-admitted in August and December, 1896, and in March, 1897, and besides this, several curettings were done under gas at out-patients. In September, 1898, the parts having been quite healed for over a year, the patient was re-admitted for a plastic operation. A portion of the upper lip was turned upwards to form a new columna nasi, the shape of the nose being completely restored by that means.

He was last seen at out-patients in June, 1899, when there was no sign of disease.

CASE IV.—The notes of this case have been kindly sent to me by Mr. Symonds. A girl of eighteen came with nasal obstruction and discharge. On examination, both inferior turbinals were enlarged and presented lobulated granulation masses along the inferior borders. The condition was thought to be hypertrophy, but when the masses were removed it was seen that the appearance resembled that of tubercle. There were distinct yellow tubercles, and in other cases rounded granular elevations, evidently ulceration. The microscopic section showed the ordinary granuloma of tubercle. The anterior part of each bone was removed, together with the granulations. The patient presented no other signs of tubercle, either in the nose, glands, or elsewhere. She was in good health, and appeared to have none other but this local disease.

CASE V.—A. P., a married woman, æt. 34, came to the out-patient department on December 15th, 1899, for nasal obstruction, chiefly affecting the right side.

Her mother died of cancer of the uterus, her father of old age. An uncle on the mother's side died of phthisis. The remaining members of the family are all healthy.

The patient has had no serious illnesses, her only trouble being a slight cough which she has at times.

The nasal obstruction was first noticed a month before her attendance at the hospital, and soon after a small amount of discharge came from the right nostril; this discharge was usually yellow but was occasionally tinged with blood. On examination, a sessile tumour, about half an inch wide by three-quarters of an inch long could be seen on the anterior part of the right side of the nasal septum close to the floor of the nose. The tumour was firm in consistence, of a pale red colour and smooth surface, it easily bled when touched with a probe. On the anterior part of the tumour a small oval ulcer could be seen; this was quite shallow and was covered with yellow discharge. With the exception of slight enlargement of the left inferior turbinated bone the rest of the nasal cavity was normal.

The surface of the swelling was cocaineised, and a small portion, including part of the ulcerated area, removed for microscopical examination. Sections showed the growth to be of typical tuberculous structure, and on staining with carbol fuchsin numerous tubercle bacilli were found. On January 1st, 1900, the patient was admitted into Ruth ward, and on the following day the growth was removed and the base of attachment thoroughly curetted. Fifty per cent. lactic acid was applied daily for the next few days, and rapid healing of the ulcer took place.

Dr. Fawcett examined the patient's chest and found nothing abnormal, with the exception of a few crepitating râles at the inner end of the second intercostal space on the left side. This may be taken as possible evidence of phthisis, and, if this is so, the nasal disease was no doubt secondary to it.

The patient was seen on January 26th, 1900. The ulcer in the nose was then quite healed, and the patient was well in every way.

CASE VI.—R. P., æt. 54, was admitted under Mr. Brailey, on May 16th, 1899. In his family history there was nothing pointing to tuberculous disease, and his only previous illness was rheumatic fever. Three months before admission, the patient noticed a small swelling beneath the inner part of the upper lid of the left eye, and experienced a little pain. This swelling steadily and

slowly increased in size up till the time of admission, in spite of treatment by means of various applications. On admission, there was a large, hard swelling under the inner portion of the left upper lid, and extending back along the inner wall of the orbit. The skin and conjunctiva over the swelling were slightly congested. Pain and tenderness were only slight. The left eyeball was displaced slightly downwards and outwards. Ocular movements were somewhat limited, especially inwards, and there was diplopia on looking inwards. There were no nasal symptoms, and no signs of any disease elsewhere.

Operation, May 26th.—Under A. C. E. an incision was made in the upper lid over the swelling, and the tumour exposed by pushing the contents of the orbit outwards, the internal rectus being controlled by means of a silk ligature passed round it. The swelling, which was firm to the touch, was then separated from the eyeball and from the inner wall of the orbit, and was found to extend a considerable distance backwards. The growth was then removed as completely as possible, but in so doing it was found that the bone was partly destroyed, and that the growth had extended into the orbit from the lateral mass of the ethmoid. During the operation there was bleeding from the left nostril.

For the next few days there was a slight sanious discharge from the left nostril, but otherwise the subsequent course was uneventful. On May 29th I examined the nose, but owing to the large size of the inferior turbinal body, nothing could be seen above it. The mucous membrane covering the inferior turbinal, septum, and floor of the nose, was normal.

On microscopical examination of the growth, it was found to be typically tuberculous in structure, there being a large number of typical giant cell systems scattered through the sections. In some parts there was also well-formed fibrous tissue. A large number of sections were stained for tubercle bacilli, but none were found.

I examined the patient again on June 16th. There was then no nasal discharge. The application of twenty per cent. cocaine produced sufficient contraction of the inferior turbinal to get a view of the parts above it. The middle turbinal was then seen to

be considerably swollen and fleshy, but there was no distinct tumour, nor was there any evidence of ulceration.

The patient was seen on January 26th, 1900. There was then no evidence of disease in the nose, and no recurrence of the tumour in the orbit could be made out.

I will now proceed to the analysis of the total of 100 cases, brief notes of which are appended in tabular form.

SEX.

Of the 100 cases, 41 were males and 59 females. Comparing this sex distribution with that of some other forms of tuberculosis it will be seen at once that the large proportion of females affected is in striking contrast to the general rule.

Dr. Sydney Martin, in his article in Clifford Allbutt's System of Medicine, on Tuberculosis, says that the disease is equally common in the two sexes. As regards phthisis, the death-rate in the two sexes is nearly equal, for taking the mean annual mortality from phthisis per 1,000,000 living at all ages for the period 1851—1880, the numbers are:—males, 2,418, females, 2,428. Again, in laryngeal tuberculosis the number of males affected is largely in excess of females, Mackenzie's figures giving—males, 365, females, 135.

AGE.

The youngest was eight months old, the oldest 71 years. The following table shows the general distribution as regards age:—

Under 1 year	1 case.
1-10 years	3 cases.
10-20	"	23 "
20-30	"	26 "
30-40	"	22 "
40-50	"	12 "
50-60	"	10 "
Over 60	"	3 "
Total					100

As will be seen, the disease is commonest between the ages of 10 and 40, and this corresponds closely with the age distribution of tuberculosis generally.

PATHOLOGY.

The disease may be either primary or it may occur in conjunction with tubercular disease elsewhere. The latter class may be designated as secondary, although it is quite possible that in some of them the disease in the nose may have started as soon as, or even before the focus elsewhere, since the progress of tubercular disease in the nose is slow and its symptoms often slight. In eight of the cases (23, 25, 50, 51, 52, 65, 66 and 95) mention is made of tubercular glands in the neck, which were either present when the case first came under observation or appeared later. As the glandular affection was in all probability secondary to the nasal disease, I have included these cases in the primary group. As an instance, Case 95 may be quoted, in which softened glands were present when the patient came for nasal treatment. The glands were removed by Mr. Symonds at the same time as the tuberculous inferior turbinal.

According to this mode of classification, 58 of the cases appear to be primary, 37 secondary, and 5 doubtful.

It is generally believed that nasal tuberculosis is nearly always secondary to tubercular disease elsewhere, and a statement to this effect appears in most of the text-books. According to my figures, 58 per cent. appear to be primary. The question arises as to whether these figures represent the truth; if they do not, what is the explanation?

In the first place, it is possible, or even probable, that some of the cases that appear to be primary are really secondary to undetected phthisis, for during life it is impossible to be certain that there is no quiescent or healed phthisis present in a given case. In my list of cases I have only included those that are fully reported, and I have only classed as primary those in which it is distinctly stated that a careful search for other tuberculous lesions had been made with a negative result.

In the second place, it might be argued that many cases occurring in patients suffering from phthisis, or other form of tuberculous disease, might be overlooked in consequence of the comparatively trivial nature of the nasal symptoms. The same argument might equally well be applied to the question of

autopsies, since it is not usual to carefully investigate the condition of the nose at post-mortem examinations, and hence a case undiagnosed during life would be equally liable to pass unnoticed at the autopsy. Reference, however, to the statistics of autopsies on tuberculous patients in which the nose was examined, throws considerable light on this point. Willigk found the nose affected once in 476 autopsies; Weichselbaum twice in 146 autopsies, and E. Fränkel in 50 autopsies found the nasal mucous membrane healthy in every instance. Thus, in 672 autopsies on patients dead of tuberculous disease, there were only three instances of tuberculous disease of the nose, or a percentage of .44. To show how very small this proportion is, I will compare the figures with some relating to laryngeal tuberculosis. In the years 1894–1897 phthisis was found in 366 post-mortems at Guy's, and in these 366 cases tuberculosis of the larynx was present 62 times. This gives a percentage of 16.9 as compared with .44 in the case of nasal tuberculosis. From these considerations it is, I think, justifiable to conclude that the proportion of primary cases is much greater than has hitherto been generally supposed.

The following table gives *the position* of the lesions :—

Septum only	70 cases.
Inferior turbinal only	5 "
Septum and inferior turbinal	6 "
Septum, floor, and inferior turbinal	6 "
Septum and floor	5 "
Septum and ala	2 "
Roof	1 "
Ethmoid	1 "
Middle turbinal	1 "
Nasal mucous membrane	3 "
Total					100

The septum is clearly by far the most common site, having been affected alone 70 times, and in conjunction with other parts 19 times, making a total of 89 per cent.

In nearly all the cases the part of the septum involved was the anterior cartilaginous portion, and frequently the cartilage was destroyed, a perforation being thus produced. Very seldom did the disease extend and involve the osseous septum, and only in one case (25) was this part of the septum alone affected.

The disease commences usually in the submucous tissue, and in a number of cases no subsequent extension either to bone or cartilage occurs.

The lesion may take the form of a swelling, ulceration, or a combination of both these. In 40 cases there was a non-ulcerated swelling, in 27 cases ulceration only without any tumour formation, and in the remaining 33 cases there was either a swelling ulcerated on the surface or a swelling in one part of the nose, together with ulceration in another part. The formation of these swellings composed of tuberculous tissue constitutes one of the most striking features of the disease, for swellings were present in no less than 73 of the cases. The surface of the swelling is usually smooth, occasionally granular; the colour is mostly described as pale red, and in a few instances as yellowish, and to the touch it feels firm and elastic. When caseation takes place the epithelium covering the surface is usually lost, and ulceration results.

In size the swellings vary from small granular areas to the size of a walnut or larger.

In Case 96 the swelling was fully one inch in diameter and completely occluded both nostrils, the cartilaginous portion of the septum having been destroyed. The most remarkable swelling is the one described by Koschier, in Case 90, in which the nasopharynx was found to be filled by a smooth, red, non-ulcerated swelling projecting from the left choana. A diagnosis of sarcoma was made. After removal, the swelling was found to have sprung from the posterior part of the left side of the septum nasi, close to the roof. On microscopical examination, the swelling was found to consist throughout of typical tuberculous granulation tissue.

In connection with this case I should like to refer again to one of Mr. Symond's cases, No. 95. The patient was first admitted in April, 1893, for a naso-pharyngeal tumour, which was thought

to be a sarcoma and which was removed by means of a snare. It is stated in the report that removal was probably not complete owing to the softness and vascularity of the growth. Moreover, although the diagnosis given in the report is sarcoma, there is no definite statement that a histological examination was made. Three years later the man was re-admitted for nasal obstruction and enlarged cervical glands, and Mr. Symonds naturally supposed that there was recurrence locally and in the glands. The nasal obstruction, however, was found to be due to enlarged inferior turbinals, and the post-nasal space proved to be quite healthy. On removal of the glands and portions of the inferior turbinals both proved to be tuberculous. In the light of the later history of this case and its similarity to Case 90, is it not possible that the original growth in the naso-pharynx was not sarcomatous at all, but tuberculous?

Herzog, in a paper on this subject, states that Bresgen has expressed the opinion that swellings are not formed in the nose as the result of tuberculous disease, and that when tubercle bacilli are present in nasal swellings, they are only found in the superficial part, owing to the fact that the infection with tubercle bacilli is a secondary one. This opinion is refuted by Herzog, who points out the fact that tuberculous swellings, often of considerable size, are found, though rarely, in other parts, and he instances the larynx.

Bresgen's theory has, moreover, been clearly disproved by the result of careful microscopical examination, for it has been shown by several observers, notably by Chiari and Koschier, that these swellings are composed throughout of typical tuberculous granulation tissue, containing numerous giant cell-systems in every part.

I have examined numbers of sections of the swellings in cases 96, 99 and 100, and in none of them did I find any evidence in support of Bresgen's contention, the sections showing only the usual structure of tuberculous granulation tissue.

There is, therefore, no longer any doubt that these swellings are purely tuberculous in origin, and their frequent formation—they occurred in 73 per cent.—when the nose is affected by tuberculosis is almost peculiar to tuberculous disease in this situation.

For although in other positions masses of tuberculous granulation tissue of considerable size may be found, these masses usually replace the normal tissue of the part, as for instance, in the ends of long bones, or in the larynx, rather than give rise to definite swellings projecting beyond the normal outline of the affected organ. Perhaps the peculiar position of the lesion in a warm, moist cavity, where it is not subjected to any pressure, and in which there is no resistance to growth from surrounding structures, plays some considerable part in determining this tumour-formation.

In some of the cases, however, the lesion is a destructive one, ulceration, with little or no new formation, being the most marked feature.

The form of lesion present in any given case must clearly depend upon many factors, but of these the number of bacilli present, their virulence, and the resisting power of the individual, are probably the most important. A small number of bacilli of a low type of virulence would set up irritation in the tissues, resulting in the formation of granulation tissue and the production of a swelling, and this would clearly be augmented by a high resisting power on the part of the individual attacked.

On the other hand, under the reverse conditions it would seem probable that the bacilli or their products would be more likely to cause destruction of tissues, resulting in ulceration.

This is borne out, moreover, by reference to the cases. For out of the 40 cases in which a non-ulcerated swelling was present, no less than 33 occurred in primary cases, and as only 56 per cent. of the cases were primary, it is clear that swellings are much commoner in primary cases and also that ulceration is commoner in secondary cases, in which several foci of disease are present, and the resisting power therefore slight. The histology of the swellings and ulcers does not call for any special description since it in no way differs from that of other tuberculous lesions. Tubercle bacilli are often very difficult to demonstrate in the sections, and often cannot be found, even after a most careful search. As would be expected, bacilli have been more frequently found in the more rapid ulcerating cases than in the more slowly

growing tumour formations. I have stained large numbers of sections of the swellings, in Cases 96 and 99, for tubercle bacilli, but was not able to find any bacilli in any of the sections. In sections from a portion of the growth near the ulcerated surface in Case 100, however, I found numbers of tubercle bacilli.

SYMPTOMS.

In nearly all the cases the chief symptom complained of by the patient was nasal obstruction, in many of the cases there was a discharge in addition to this. In the cases in which there was chiefly ulceration without marked tumour formation, the nasal obstruction was often due to the formation of crusts in the nostrils, composed of inspissated discharge.

The discharge is usually described as muco-purulent or purulent, and at times blood stained.

After removing any discharge and crusts that are present in the nostrils, examination reveals a pinkish sessile tumour, which may be ulcerated on the surface, or else a red infiltrated area which is the seat of one or more shallow ulcers, with uneven bases, covered with yellow discharge or dried crusts. The tumours and infiltrated areas, as well as the surrounding mucous membrane, are generally markedly vascular and easily bleed when touched with a probe. The staining of the discharge with blood is probably caused by injury inflicted on these vascular growths by attempts to clear the nostrils.

The symptoms above described are those usually present, but in some cases there may be special symptoms, dependent upon the form, position, and size of the lesion present.

For instance, in Cases 90 and 91 the disease gave rise to a swelling projecting into the nasopharynx, and the symptoms were those of nasopharyngeal tumours generally, the nature of the growth having being discovered only after removal. Case 100 was a very exceptional one, the disease having started in the lateral mass of the ethmoid. This case presented itself as one of intra-orbital tumour and not as one of nasal disease at all.

DIAGNOSIS.

It is unnecessary here to enter fully into the question of the differential diagnosis of intra-nasal tumours and ulcerations generally. I shall accordingly simply lay stress on the special points

in the diagnosis of this disease which have occurred to me in studying the cases.

In the reports of cases frequent mention is made of mistakes in diagnosis. Where such mistakes have arisen the disease has usually been thought to be either a tertiary syphilitic lesion or a malignant growth.

Apart from the presence of other evidences of syphilis, gummatous disease of the interior of the nose is commonly much more extensive and destructive than tubercular disease. It is also, as a rule, early associated with necrosis, as is evidenced by the peculiar fœtor which is commonly present.

Where, however, a single gumma is present without ulceration it may be impossible to diagnose it at sight from a tuberculous lesion, and in such cases only the result of treatment and of the histological examination of a portion of the growth can clear up the difficulty.

With regard to the question of a malignant growth, it must often be quite impossible to make a correct diagnosis from history and appearances only. The nasopharyngeal tumours in Cases 90 and 91, and the intra-orbital tumour in Case 100, at once again occur to one as good instances of the extreme difficulty that may arise in attempting to make a correct diagnosis. Apart from such exceptional cases as these, however, a simple tumour on the septum or inferior turbinated body may be quite as difficult to diagnose.

The only certain means of arriving at a correct conclusion is to remove a small portion of the growth under cocaine anæsthesia and to examine it microscopically, and also, if the means are at hand, to inoculate a guinea-pig.

In the ulcerative type of the affection, in which on examination the walls of the nasal cavity are found to be covered with dried crusts, the disease has been mistaken for atrophic rhinitis. Careful cleansing and removal of the crusts will, however, remove doubt by demonstrating the presence of ulceration in a case of tuberculous disease, whereas in atrophic rhinitis the epithelial surface will be found to be intact.

TREATMENT.

This in no way differs from that of other forms of tuberculosis, and consists essentially in the complete removal, wherever possible, of the whole of the diseased tissues. The details of the following account of technique and indications are gathered from those given in the reports of the various cases.

The only exceptions to the rule of complete removal are cases in which there is extensive phthisis, or tuberculous disease in some other part of the body, owing to which the patient is too ill to undergo an operation. Even though there may be a considerable amount of phthisis, provided that the patient is not too ill, the operation should still be performed, for the removal of one focus of disease will necessarily give the patient a better chance of successfully combatting the others.

Should a swelling be present, the main portion may be removed with the knife, or, where possible, with the cold snare. The base of attachment should then be carefully and thoroughly curetted, and any undermined portions of mucous membrane cut away. Bleeding is likely to be free, but it usually quickly stops if pressure is applied for a short time. When bleeding has ceased, the surface should be either cauterized thoroughly with the actual cautery, or pure carbolic acid rubbed in, care being taken to limit the application of the acid to the affected area. If the disease consists of an ulcerated area, this should be curetted and cauterized in the same way.

In the cases in which one of the turbinals is the seat of disease, as much as is necessary of the turbinal should be removed with scissors, or the spokeshave. During healing, the nose should be kept free from discharge and crusts by means of an alkaline lotion, or spray, and the ulcer may be stimulated by the application of fifty per cent., or stronger, solution of lactic acid.

Owing to the difficulty in getting rid of the deeper part of the diseased tissue where it is invading the healthy structures, frequent recurrences have taken place. This necessitates repeated curetting and cauterization; but if this can be done, and the patient can be kept under observation for a sufficient length of time, a complete cure can generally be obtained. Case 97 is a

good example of the inveterate tendency to recur, and also of the final success of persistent treatment. The patient, who had a swelling of the septum, was operated on in October, 1895, August and December, 1896, and on several occasions in 1897. No further recurrence took place after September, 1897, and when last seen in June, 1899, there was no sign of disease.

APPENDIX OF CASES.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
1	Laveran ...	M.	25	Secondary	Septum ...	Ulceration ...	Phthisis ...	Died of pneumo- thorax
2	" ...	M.	43	Secondary	Septum ...	Ulceration ...	Phthisis ...	Died of phthisis
3	Reidel ...	M.	63	Primary ...	Septum ...	Tumour ulcerated on surface Perforation of septum	None ...	Recurred after treatment
4	" ...	F.	55	Primary ...	Septum ...	Tumour 1½ cm. long, ulcerated on surface. Perforation of septum	None ...	—
5	Spillman ...	F.	33	Secondary	Septum ...	Ulceration ...	Phthisis ...	—
6	Thornwald ...	M.	26	Secondary	Septum, left inferior turbinal, floor of nose	Tumours, ulcerated on the surface	Tuberculosis of pharynx, larynx and lungs	—
7	Riehl ...	M.	36	Secondary	Left side of septum	Ulceration ...	Phthisis ...	Died of phthisis
8	Demme ...	M.	1	Secondary	Septum ...	Ulcer and small nodules	Tuberculosis of lungs	Died aged 1½ years

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
9	Demme ...	M.	8 mos.	Primary ...	Wall of nose ...	Nodules and ulcer	None when first seen	Died of tuberculous meningitis
10	Sokolowski	F.	20	Secondary	Septum and floor of nose	Ulcer with a tumour on anterior margin	Tuberculosis of larynx and lungs	Died of phthisis
11	Schaefer	F.	51	Primary ...	Septum ...	Tumour ...	None ...	Recurrence
12	"	F.	26	Primary ...	Septum ...	Tumour ...	None ...	Removal, recurrence
13	"	F.	39	Primary ...	Septum ...	Tumour ...	None ...	Removal, recurrence
14	"	M.	32	Primary ...	Septum ...	Tumour ...	None ...	Recurred several times
15	"	M.	57	Primary ...	Septum ...	Tumour ...	None ...	Well 9 months later
16	"	M.	51	Primary ...	Septum ...	Tumour ...	None ...	Recurred
17	Cartaz ...	M.	42	Secondary	Septum ...	Ulceration ...	Phthisis	Died of phthisis
18	Bruns ...	M.	30	Primary ...	Septum ...	Tumour, ulcerated on the surface	None ...	Recurrence 5 months after removal

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
19	Michelson	F.	27	Primary ...	Septum ...	Tumour	None ...	Ulceration after removal
20	"	F.	21	Secondary	Septum ...	Ulceration	Tuberculosis of larynx and lungs	Died of phthisis
21	"	F.	19	Secondary	Left inferior turbinal	Tumour	Tuberculosis of larynx and lungs	—
22	"	F.	48	Primary ...	Septum ...	Tumour	None ...	—
23	"	M.	18	Secondary	Septum and ala nasi	Ulceration	Tuberculosis of upper lip and cervical glands	—
24	Hajeck	M.	30	Secondary	Septum ...	Ulceration	Phthisis ...	Ulceration after removal
25	"	M.	13	Primary ...	Septum osseum...	Ulceration	Tuberculosis of cervical glands	—
26	"	F.	24	Primary ...	Septum ...	Ulceration	None ..	—
27	Mertens	F.	53	Primary ...	Right inferior turbinal, left side of septum	Ulceration	None ...	—

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
28	Beermann	F.	34	Primary ..	Right side of septum	Small tumour	None ..	Recurred after 3 months
29	Hahn	F.	22	Primary ..	Septum and right inferior turbinal	Small tumour	None ..	No recurrence 6 months later
30	"	F.	55	Primary ..	Septum ...	Tumour as large as a hazel nut	None ..	Removal. No recurrence 3 months later
31	"	F.	17	Primary ..	Septum ...	Two small tumours	None ..	—
32	"	F.	19	Primary ..	Septum ...	Ulcer ..	Blepharitis, keratitis	—
33	"	F.	17	Secondary	Septum ...	Ulcer and granular masses	Dulness over left apex	—
34	Fick	F.	26	Secondary	Left side of septum	Ulcer ...	Tuberculosis of nasal duct. Suspicious apices	—
35	Jurasz	F.	19	Secondary	Right side of septum	Tumour ulcerated on the surface	Tuberculosis of skin of left thumb and right cheek	Died of phthisis 1 year later

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
36	Jurasz ...	M.	36	Secondary	Left side of septum	Ulcer ...	Tuberculosis of lungs, ribs and buccal mucous membrane	—
37	" ...	M.	44	Secondary	Right side of septum	Ulcer ...	Tuberculosis of lungs, larynx and tongue	—
38	Olympites ...	M.	36	Secondary	Septum ...	Ulcer ...	Tuberculosis of lungs and larynx	—
39	Moure ...	M.	29	Secondary	Right side of septum	Ulcer ...	Tuberculosis of lungs and larynx	—
40	Rault ...	M.	35	Secondary	Right side of septum	Ulcer ...	Tuberculosis of lungs	—
41	" ...	F.	40	Secondary	Right side of septum	Tumour	Tuberculosis of larynx and lungs	Tumour removed. Died 18 months later of phthisis

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
42	Rault ...	F.	23	Primary ...	Left side of septum	Tumour ...	None ...	Recurred four times after removal. Well 5 months after last operation
43	Capart ...	F.	25	Primary ...	Right side of septum	Tumour ...	None ...	Removal. No recurrence
44	" ...	F.	53	Probably Secondary	Right side of septum	Tumour ...	—	Died of miliary tuberculosis
45	" ...	M.	35	?	Septum ...	Tumour ...	—	—
46	Vincenzo Cozzolius	F.	Young girl	Secondary	Septum ...	Ulcer with perforation	Tuberculosis of lungs	Died of phthisis
47	Baber ...	F.	18	Secondary	Septum ...	Ulcer with perforation	Strumous disease of upper air passages	—
48	Browne ...	F.	20	Secondary	Nasal mucous membrane	Ulceration ...	Tuberculosis of pharynx and larynx	—
49	Fitzpatrick ...	M.	25	Secondary	Nasal mucous membrane	Ulceration ...	Tuberculosis of pharynx and larynx	Died of phthisis

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
50	Herzog ...	F.	24	Primary ...	Left inferior turbinal	Granulation, later large tumour	Dacryocysto-blennorrhoea. Suppurating cervical glands	Several recurrences after removal. Later glands removed and found to be tuberculous
51	" ...	M.	12	Primary ...	Right inferior turbinal, floor, and septum	Ulceration, perforation of septum	Enlarged cervical glands	Result good after 3½ years' treatment
52	" ...	F.	26	Primary ...	Inferior turbinal and septum	Tumour and ulceration	Vestiges of glandular suppuration	Healed in 1 month
53	" ...	M.	50	?	Right middle turbinal	Tumour ...	Dulness over right apex	Cured
54	" ...	M.	31	Primary ...	Left side of septum	Tumour and ulceration	None ...	—
55	" ...	F.	49	Primary ...	Right ala and septum. Later floor and inferior turbinal	Ulceration, perforation of septum	Dacryocysto-blennorrhoea	Finally healed in 6 months
56	" ...	M.	21	Primary ...	Inferior turbinal	Tumour ...	None ...	—

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
57	Herzog ...	M.	33	Secondary	Right side of septum	Ulceration ...	Tuberculosis of lungs and larynx	Healed in 1 month
58	C. F. Theisen ...	M.	36	Primary ...	Left side of septum	Tumour ulcerated on surface	None ...	Well 18 months later
59	Polyak ...	F.	49	Primary ...	Anterior part of septum	Tumour perforating septum	None ...	—
60	Farlow ...	F.	23	Primary ...	Left side of septum	Ulceration ...	None ...	Cured
61	" ...	F.	71	Primary ...	Right side of septum	Tumour ...	None ...	—
62	St. Clair-Thomson	F.	25	Secondary	Septum and inferior turbinals	Ulcerated tumour on septum. Ulceration of turbinals	Ulceration of buccal pharynx	Improved
63	Chiari ...	M.	16	Primary ...	Septum ..	Tumour ...	None ..	—
64	" ...	F.	9	Primary ...	Right side of septum	Tumour, later perforation	None ...	Finally cured

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
65	Chieri ...	F.	42	Primary ...	Both sides of septum	Ulceration and nodular masses	Enlarged glands in the neck	—
66	" ...	F.	12	Primary ...	Left side of septum	Pale yellow tumour	Enlarged glands in the neck	Cured after several curettings
67	" ...	M.	29	?	Posterior third of right septum	Pale tumour ...	? Tubercle	Died a year later of miliary tuberculosis
68	" ...	M.	40	?	Turbinals and septum	Nodular masses	Lungs not examined	—
69	Pluder ...	M.	34	Secondary	Septum and floor	Infiltration ...	Tuberculosis of larynx and lungs	—
70	Sachs ...	M.	11	Primary ...	Left side of septum	Tumour ...	None ...	—
71	" ...	F.	23	Primary ...	Left side of septum	Tumour size of a hazelnut	None ...	—
72	Baurowicz ...	F.	40	Primary ...	Septum ...	Tumour ...	None ...	—

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
73	Baurowicz	F.	14	Primary	Septum	Tumour	None	—
74	"	F.	24	Primary	Septum	Ulcerated tumour, septum perforated	None	Cured
75	Wroblewski	M.	20	Primary	Septum	Tumour	None	Finally cured. Frequently curetted
76	"	F.	41	Secondary	Septum and right inferior turbinal	Ulcer on septum Tumour on turbinal	Tubercular otitis	Repeatedly removed, recovery not complete
77	Sachs	M.	14	Primary	Septum	Tumour as big as a cherry	None	—
78	Koschier	F.	35	Secondary	Septum, floor and inferior turbinal	Extensive infiltration, ulceration in two places	Tuberculosis of larynx and lungs	Disease spread in spite of treatment
79	"	M.	36	Secondary	Right side of septum	Tumour ulcerated on the surface	Tuberculosis of larynx and lungs	Improved

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
80	Koschier	M.	38	Secondary	Septum and floor of nose	Infiltrated area with ulcerated surface	Tuberculosis of both lungs	Did not heal in spite of treatment
81	"	F.	48	Secondary	Both sides of septum	Tumour size of half a walnut	Signs in apices. Tubercle bacilli in sputum	Cured
82	"	M.	7	Primary ...	Left side of septum	Tumour with ulcerated surface	None	Recurred 7 months after removal
83	"	F.	50	Secondary	Both sides of septum	Large tumour ulcerated on one side	Tuberculosis of lungs	Healed rapidly after curetting
84	"	F.	53	Primary ...	Left side of septum and floor	Infiltration with ulcers on surface	None	Recurred after treatment. Patient well 6 months after second curetting
85	"	F.	25	Primary ...	Left side of septum	Tumour	None	Well one year after treatment

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
86	Koschier	F.	43	Primary ...	Both sides of septum	Tumours	None ...	Well 1 year later
87	"	F.	71	Primary ...	Right side of septum, left side affected later	Infiltrated area with many small ulcers on the surface	None ...	A tumour had been removed from same site 20 years before The disease was curetted away, but recurred a year later. Again curetted, well 6 months later
88	"	F.	25	Secondary	Left side of septum	Tumour	Dulness over both apices	Healed
89	"	F.	15	Primary ...	Right side of septum	Tumour	None ...	Healed, no recurrence
90	"	F.	20	Primary ...	Left side of septum	Smooth non-ulcerated tumour, size of a small apple, projecting through left choana into naso-pharynx	None ...	Removed, no recurrence 3 months later

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
91	Koschier	F.	19	Primary ...	Back part of roof, close to septum on right side	Smooth non-ulcerated tumour, size of a date, projecting into nasopharynx	None ...	Healed quickly after removal
92	"	F.	22	Primary ...	Septum, floor and both turbinals	Large tumour blocking both nostrils. No ulceration on septal tumour, ulcers on floor and both turbinals	None ...	Patient died a year later of phthisis and tuberculous meningitis
93	"	F.	54	Secondary	Both sides of septum and floor	Ulceration with infiltrated margins	Dulness and rales over both apices	Healed rapidly
94	"	F.	22	Primary ...	Septum, floor and inferior turbinal on right side	General infiltration, ulcerated in places	None ...	Cured

APPENDIX OF CASES—continued.

Number.	Surgeon.	Sex.	Age.	Primary or Secondary.	Site of Lesion.	Form of Lesion.	Disease elsewhere.	Result.
95	Symonds	M.	36	Primary ...	Right inferior turbinal	Tumour ...	? Fibro-sarcoma of nasopharynx 3 years before. Later tuberculous glands in neck	Cured, well 5 years later
96	"	F.	35	Primary ...	Both sides of septum	Tumour, size of a walnut, slightly ulcerated	None ...	Cured, well 2 years later
97	"	M.	17	Primary ...	Right side of septum	Ulcerated tumour	None ...	Frequently cured during 3 years. Finally cured
98	"	F.	18	Primary ...	Anterior ends of both inferior turbinals	Points of caseation and ulcerated areas	None ...	Cured
99	"	F.	34	? Secondary	Septum ...	Ulcerated tumour	Slight signs over left apex	Well 1 month later
100	Brailey	M.	54	Primary ...	Left lateral mass of ethmoid perforating os planum	Tumour ...	None ...	Well 6 months later

BIBLIOGRAPHY.

-
1. Morgagni, 'De Sedibus et Causis Morborum,' 1765.
 2. Willigk, 'Prager Viertel-jahrschrift,' xxxviii., p. 4.
 3. Laveran, 'Union Medicale,' 1877, Nos. 35, 36.
 4. Volkmann, 'Sammlung Klinischer Vorträge.,' Leipzig, 1879, No. 168, p. 31.
 5. Thornwald, 'Deutsches Archiv. für Klin. Med.,' Bd. xxvii., p. 586.
 6. Weichselbaum, 'Allgemeine. Wien. Med.,' Zeitung, 1881, Nos. 27, 28.
 7. Riehl, 'Wiener. Mediz. Woch.,' 1881, No. 45.
 8. Demme, 'Berlin. Klin. Woch.,' 1883, p. 218.
 9. Sokolowiski, 'Beitz. Chir. v. Bruns.,' Baud. iii., fasc. 3, p. 438.
 10. Schafer, 'Deutsche Med. Woch.,' 1887, No. 16.
 11. Bresgeu, 'Deutsche Med. Woch.,' 1887, Nos. 20, 22, 27.
 12. Cartaz, 'De la Tubercul. Nasale.,' Paris, 1887.
 13. Bruns, 'Bruns' Beitr. z. Chir.,' Bd. iii., fs. 3, p. 433.
 14. Merteus, 'Dissert. Inaug. Wuerzburg,' 1889.
 15. Hajeck, 'Inter. Klin. Rundschau,' 1889.
 16. Michelzohn, 'Zeitschr. f. Klin. Med.,' 1890, Bd. xvii.
 17. Beermann, 'Dissert. Inaug. Wuerzburg,' 1890.
 18. Hahn, 'Deutsche Med. Woch.,' 1890, No. 13.
 19. Fick, 'Correspond-blatt. f. Schweiz.,' Aerzte, 1891, p. 211.
 20. Jurasz, 'Die Krankheiten der Obern Leftwege.,' Heidelberg, 1891.
 21. Olympites, 'Thèse de Paris,' 1890.
 22. Moure, 'Thèse de Paris de Boutard,' 1869.
 23. Rault, 'Thèse de Paris de Boutard,' 1889.
 24. Capart, 'Bull. de l'Academie Roy. de Med. de Belg.,' 1890.
 25. Vincenzo Cozollino, 'Gaz. Medic. di Roma,' 1889.
 26. Baber, 'B. M. J.,' 1889, No. 22, p. 1407.
 27. Browne, 'Sajous Annual,' 1888, vol. iii.
 28. Fitzpatrick, 'Cni. Lancet Clinic,' 1891, p. 468.
 29. Herzog, 'American Journal,' 1893.
 30. Farlow, 'Report of American Laryngol. Assn.,' 1893.
 31. Strauss, 'Münchener. Med. Woch.,' 1894, No. 28.
 32. Wroblewski, 'Internat. Centrallbl.,' July, 1894, s. 16.
 33. Manasse, 'Archiv. f. Path., Anat. u. Physiol.,' Band. 147, Heft 1, s. 23.
 34. Koschier, 'Wiener. Med. Woch.,' 1895, No. 36.
 35. Pluder, 'Münchener. Med. Woch.,' 1895, No. 45.
 36. Strauss, 'Münchener. Med. Woch.,' 1894, No. 28.
 37. Baurowics, 'Internat. Centrallbl.,' Sept., 1895.
 38. Chiari, 'Archiv. f. Laryngol.,' Bd. 1, Heft. 2.
 39. Panzer, 'Wiener. Med. Woch.,' 1895.

40. Gourdiat, 'Rev. d. Mal. Cancer.,' 1896.
41. Gerszewski, 'Ueber Tubercul. d. Nase,' 1896.
42. Schmidt, 'Die Krankheiten d. Oberen Luftwege, 1897.
43. Gaudier, 'Tumeur des Fosses Nasaes.' Nord. Méd., March 15th, 1897.
44. Sachs, 'Münchener. Med. Woch.,' 1897.
45. Sachs, 'Deutsche Med. Woch.,' 1896, No. 4.
46. Polyak, 'Med. News,' May, 1896.
47. Hicquet, 'Internat. Centrallbl.,' Aug., 1896, s. 370.
48. Boluminski, 'Beiträge. z. Tuberculose d. Oberen. Luftwege,' (Gerber's Clinic., 1895).
49. St. Clair Thompson, 'Brit. Med. Journ.,' 1897, ii., 1263.
50. Theisen, 'Laryngoscope,' Feb., 1898.

A CASE OF RECURRENCE OF CALCULUS IN A PARAPLEGIC SUBJECT— SUPRAPUBIC LITHOTOMY—TWICE PERFORMED.

By JOHN MILLS THORN.

J. H., æt. 61. In early life a plumber. Some thirty-eight years ago, after getting very cold and wet when working one day down a well repairing a pump, he became during the following night paralysed, and was seriously ill for some months, since which time he has suffered from paraplegia. For the last ten years he has been obliged to use a catheter continually for drawing off his urine, and lately the urine has been getting very foul; sometimes he has to pass the catheter every half-hour, and at night he usually holds it in the bladder and allows it to drain into a jar.

Up to three years ago he used to assist his father, a butcher, and drove about daily. He had to relinquish the driving about as he felt he was becoming weaker, and at any time he was subject to very severe and sudden attacks of pain, the jolting of the cart especially being likely to cause an attack.

In September, 1896, I was called in to see him for the first time. He was suffering from intense pain in the left eye, left side of nose and forehead. A day or two later well-marked herpes frontalis was developed, which ran a very tedious and painful course; there was acute conjunctival inflammation, and a small ulcer of cornea developed, which left a slight opacity.

During this attack the condition of the urine was very foul, with much mucus and sediment, leaving one to believe that he was suffering from cystitis and probably ascending nephritis. At times his legs and thighs were jerked up and down and kicked about in any direction by clonic spasms, entirely involuntary; at the same time there was frequently spasm of the urethra, and great difficulty in introducing a catheter; occasionally he had a few days free from pain, and then, perhaps instantly, he was thrown into acute agony for hours, or days, with all the attendant troubles of the spasms and rigidity in legs and thighs, &c.

For nearly ten months his bladder was washed out with Sir. H. Thompson's fluid twice a day; this prevented the urine becoming more foul, but by no means kept it sweet.

In August, 1897, while passing a soft *coudée* catheter he believed that he touched a stone. I also came to that opinion, and attempted, unsuccessfully, to introduce a sound (Buxton Brown's), owing to there being prostatic enlargement; however, with a well-curved metal catheter, the fact of there being a stone in the bladder was assured.

His condition was very miserable, and in spite of the risk, it was decided to operate by suprapubic lithotomy.

He was admitted into the private room at the Retford Hospital, and on 7th October, 1897, anæsthetised (A.C.E.); the bladder was distended with warm boracic lotion, about 12 ounces syringed in through a well-curved metal catheter, which was tied in the penis, and used as a guide in opening the bladder.

There was then no difficulty in reaching the bladder, and a calculus extracted which weighed $1\frac{3}{4}$ ounces; it was smooth and constricted in middle of its long diameter, which made it figure of eight (8) in shape.

The lining of the bladder was much inflamed, congested, and there was a quantity of muco-pus coating it. Boracic lotion was freely used to irrigate with. It was decided to introduce a soft catheter through the urethra, and tie it in (this was with much difficulty performed), and then to suture the bladder. A continuous suture of fine silk was used for the mucous membrane,

and Lembert sutures, four or five, for the muscular wall of the bladder. After these had been tied the bladder was injected with a little lotion, and no fluid escaped through the sutured wound. The abdominal wall was then united with silkworm gut sutures, a small drain of iodoform gauze inserted, and the wound dressed with iodoform gauze. He was removed to bed and the catheter was connected by rubber tubing with a basin of lotion set at a lower level than the bed, and acting as a syphon.

For the first three days the patient suffered from constant vomiting and sleeplessness. He noticed that he was free from pain, and there was entire absence of the spasms and involuntary movements of the lower limbs.

The bladder was irrigated two or three times daily with small quantities of warm boracic lotion; there was some hæmorrhage the first day, and shreds of mucus, which during the following days became fewer; the urine also became less foul.

Six days after the operation the catheter became blocked, which caused the bladder to be distended and very painful. The catheter was removed, but a fresh one could not be passed for several hours. Some urine forced its way through the wound, and escaped by that route; afterwards, when a catheter was passed, the urine continued to escape through the wound. A day or two later a Jacques's catheter was passed through the abdominal wound into the bladder, and stitched to the edges of the wound. The tubing that had been used previously was connected again, and for several days the urine was syphoned away through the catheter in the wound, and little or none escaped into the dressings or bed, thus keeping him free from the annoyance of a wet bed and the constant changing of draw-sheets, &c.

About twelve days from the operation, a catheter was passed per urethram, and acted well; the Jacques's catheter was removed from the wound and he made a very satisfactory recovery, leaving the hospital in nineteen days from the operation.

After being home for ten days there was a slight balanitis caused by irritation of the sutures which had fixed the catheters

in the urethra ; the prepuce was incised, and he had no further trouble.

From this time onward he was free from pain ; there were no spasmodic involuntary movements of the lower limbs, the urine was sweet ; he noticed that a flexible catheter lasted very much longer than it used to do when the urine was foul ; he required to pass the catheter only once or twice during the night : the bladder was irrigated daily with Sir Henry Thompson's fluid, and he had no difficulty in passing a catheter.

At Christmas time he was in very good health and had put on flesh.

About the middle of January, 1898, he had pain in his back for several days, and then in lower part of abdomen ; he became again subject to spasms of the lower limbs ; the urine was cloudy and not sweet ; frequent desire to micturate was present.

Soon after this he detected a stone by means of his flexible coudée catheter while drawing off his urine ; a few days later I confirmed his diagnosis.

His pain was quite as severe as on the former occasion, and the nights were very disturbed ; the urine was not nearly as foul as it was before the operation ; he was also in better health than at that time, although the deterioration of his health after the symptoms of stone appeared again was very marked indeed when contrasted with his condition at Christmas time.

An attempt to introduce a Fergusson's Calculus Extractor without an anæsthetic was unsuccessful. He then desired to have an operation performed as before. He was admitted into a private room at the Retford Hospital, and on March 9th, 1898, an anæsthetic (A.C.E.) was administered. The calculus extractor was with difficulty introduced, owing to prostatic enlargement. Then a stone was easily grasped but could not be extracted, only a few grains of sand coming away on the blades of the instrument. A second attempt to pass the instrument failed, and so too with a metal catheter. With some difficulty a soft coudée catheter, No. 10, was introduced and tied in the bladder, this was distended with warm boracic lotion by a syringe, and the bladder reached by making the incision through

the old scar in the abdomen. On opening the bladder three stones were extracted, weighing together 3jss., very friable and rough. While looking at them, just removed, it was noticed that in two of the calculi, or fragments, there was a piece of fine silk which formed the nucleus upon which the calculus was deposited. This decided me against using again a suture of silk for the mucous membrane and bladder wall, as doubtless the silk in the calculus was the piece used for the sutures in the former operation.

Owing to the thickness of the abdominal wall, it was found very difficult to use a suture of catgut; the bladder was therefore fixed with salmon gut sutures to the abdominal wall, and the urine allowed to escape through the wound. Powdered boracic acid was *freely* dusted over wound, with a little iodoform. Boracic dressings, with wood-wool, were employed, and were changed two or three times a day.

There was, as on the former occasion, very much sickness and nausea for about three days after the operation; the man was not able to rest well; the spasmodic movements of lower limbs were on this occasion present, and caused him a great deal of annoyance; the soaked sheet, in spite of being frequently changed, added to his discomfort. A small sore formed on the left buttock but happily did not spread.

At the end of a week a catheter was introduced through the urethra, and connected with tubing so as to act as a syphon, but little or no urine came by this route.

Ten days after the operation, in the evening, he was wandering mentally, and there was less urine escaping; a Jacques's catheter was introduced through the wound into the bladder, and used, as on former occasions, for a few days; the slight delirium, happily, quite cleared up and showed no sign of recurring.

A fortnight after the operation a Jacques's catheter was passed through the urethra and tied in the bladder; this carried off the bulk of the urine, but some escaped through the wound until the nineteenth day after the operation. He was removed to his home, in an ambulance, twenty-one days after the operation.

Drs. Trevor Pritchard, Hooker, and Bollands, of Retford, gave me great assistance at the operations, and, on the first occasion, we had the benefit of Dr. Norburn's counsel and aid.

POINTS OF INTEREST.

1. The calculi, or three fragments of calculus extracted at second operation. I believe that when grasping the calculus with the Fergusson's Extractor it (being very friable) was broken into three fragments, and in the fractured ends was seen the fine silk in the centre, almost exactly like the thread in middle of a piece of sugar-candy. It is of interest as giving fairly exact data as to the rate of increase of a calculus in a given known time.

The calculi have been sent to Guy's Hospital Museum.

2. The risk of using buried silk sutures for the mucous membrane and wall of bladder in cases where the urine is not sterile. Possibly, catgut if used would be free from this risk.

It occurred to me since that sutures of salmon-gut, passed through the edge of wound of bladder and brought out through skin of abdomen, in the same manner as often used to suture wound of wall in abdominal section, might be tried; as, if there arose any bad symptom, the sutures could be cut and the wound allowed to gape freely. From my experience of this one case it is very much to the patient's comfort to be saved from having a constantly wet bed for ten days or more, and makes it worth while to attempt to suture the bladder when possible.

3. After the second operation, boracic acid powder was very thickly dusted over the wound each time it was dressed; in fact, the urine made it into lotion; the skin around the wound all along kept healthy and unirritated, much more than if some more powerful and irritating antiseptic dressing had been used.

4. That even in an unfavourable case, as here in a paraplegic patient, the removal of the calculus causing intense pain and trouble may be followed by such very marked benefit and relief as to make one feel justified in attempting an operation, although the risk is fully acknowledged to be great.

5. The soundness of the patient's constitution, in spite of many years of paralysis and ten years of "catheter life," aided by the manful pluck with which he faced two severe operations, and his fortitude during his recoveries, makes me hope that it will please God to grant him a long spell of complete relief and freedom from pain and discomfort.



ON SOME CASES OF TUBERCULOUS BURSITIS.

By C. H. FAGGE, M.S.

THE importance and occasional obscurity of tubercular disease, the little that is known about Bursitis of a tubercular nature, and the need of being able to differentiate it from disease of the closely contiguous joints, will be sufficient reasons for my having collected below notes of several cases which came under my notice as Surgical Registrar, and which seem to me to illustrate clearly several points in the diagnosis of the lesions of bursæ near joints, or over bones, from actual disease of the joints or bones themselves.

Though chiefly interested in the question of diagnosis, I shall attempt, as far as these few cases serve, to draw attention also to the prognosis and treatment of this disease in its many different situations. For my purpose two kinds of bursæ may be distinguished (*a*) those connected with joint cavities, either constantly or as a recognized anatomical variation ; and (*b*) those placed over or near joints, or around some tendon at its bony insertion. Disease of the former class cannot be considered to be a pathological entity apart from the disease in the adjacent joint, and is in fact only symptomatic, as is well exemplified in the very common distension of the semi-membranosus bursa with chronic synovitis of the knee-joint.

It is therefore entirely with the second class of bursa that this paper will deal.

Of recent years some light has been shed on the darkness which covered the pathology of the many different lesions classed under chronic bursitis, and traumatism as the cause has given way to tuberculosis, syphilis or gonorrhœa, owing to more

detailed microscopical examinations demonstrating either histological changes characteristic of these several diseases, or, more definitely still, in some of them by the recognition of the presence of the bacillus, which is known to be the cause of pathological changes in the tissues.

With regard to tuberculosis of these bursæ, two forms are to be recognised clinically which exactly reproduce the pathological classification introduced by Duplay and Reclus for tuberculous teno-synovitis, viz. :—

- a.* Bursal hygroma with or without the so-called melon-seed bodies.
- b.* Fungating bursitis, in which the cavity is lined with vascular granulation tissue, forming a pulpy tumour without much secretion of synovial fluid.

Lejars¹ has further subdivided the disease into the following pathological forms :—

- a.* Serous bursitis
- b.* Fungous bursitis
- c.* Cold abscess
- d.* Serous with melon-seed bodies (*l'hygroma à grains riziformes*)
- e.* Myxomatous bursitis

and quotes a case of the first-named variety affecting the bursa over the great trochanter.

The course of the two clinical varieties above noticed is widely different, the latter being of shorter duration, associated with more acute inflammatory signs, and frequently by suppuration ending in a sinus which leads into an irregular cavity lined by a pyogenic membrane which it is often difficult to recognise as an altered bursal wall.

No doubt the difference of the behaviour in these two classes is primarily due to the virulence of the organism as opposed to the resistance of the tissues; and when the nutrition of bursal tissues is taken into account, it is not surprising that tuberculous disease here generally takes this more virulent fungating form.

¹ *Revue de la Tuberculose*, 1893, vol. i., p. 103.

It is probable that the tuberculous origin of some cases of chronic bursal cystoma containing clear fluid (bursal hydroma) is often missed, and that the weaker attack of the bacillus on the tissues in this variety is easily overcome by one or moreappings, for in this relation we seem to have an exact parallel in the behaviour of the mild ascitic form of tuberculous peritonitis, as opposed to the more rapidly fatal and intractable ulcerous variety.

Again, it is in the fungating form of tuberculous bursitis that extension to the subjacent joint or bone occurs as an occasional complication, as certainly one of my cases shows, but there seems to be no evidence to suggest that the more chronic form has this evil tendency.

That the bursal lesion is primary, and not secondary to joint or bone disease, is of course sometimes difficult to prove, especially in the second case, where the adjacent bone might be regarded as the original focus, of which the differential symptoms may be comparatively quiescent—so I have been careful to deal only with cases in the notes of which it is expressly stated that there was no evidence of disease of the bone or joint, by the closest inspection and manipulation of the parts during operation.

The evidence as to the tuberculous nature of these cases is not as complete as I had wished, for when I began microscopical examination of sections of the bursal walls of the three cases in which I was able to obtain fragments for section, I expected to be able to demonstrate in at least a majority the presence of tubercle bacilli. The number of cases is of course small, yet in none have I found any bacilli, though in each a large number of slides were examined after staining by the Ziehl-Neelsen or Gabbet's method.

On the other hand, there is in all the cases to be seen typical tuberculous granulation tissue, containing besides multinucleated giant cells, epithelioid and inflammatory cells, appearances which in the minds of some are sufficient evidence of tuberculous origin.

I am inclined, from a frequent failure to find tubercle bacilli in suitably stained sections of several other tissues besides bursæ, *e.g.*, glands, to agree with those who think that this confirmatory evidence is often difficult to obtain, and failure has no positive

value, especially when, as in two of my three bursal cases, suppuration, due to pyogenic cocci, had much increased the degeneration of the tuberculous granulation tissue.

The literature to hand on this subject confirms Arbuthnot Lane's² statement that tuberculous bursitis most commonly affects the gluteal bursa which lies between the gluteus maximus and the great trochanter.

A case of tuberculous disease in this position is a very suitable one as illustrating primary disease of this structure, for this bursa has never been demonstrated to communicate with the synovial cavity of the hip-joint, so that it seems impossible that disease of the bursa should be secondary to disease of the adjacent joint; moreover, in the cases I have collected I have been unable to find one in which secondary involvement of the bursa followed on tuberculous disease of the epiphysis of the great trochanter, though Lane (*loc. cit.*) regards this as a probable cause for these bursal lesions.

Tuberculous bursitis over the great trochanter first attracts attention in the large majority of cases by the presence of a swelling which has all the characteristics of a cold abscess, situated over the great trochanter.

I have observed that the actual situation of this swelling is some help in the diagnosis from an abscess due to disease of the hip-joint, for with the latter the tumour³ most commonly comes forward from beneath the glutei in front of and below the great trochanter, near the insertion of the tensor fasciæ femoris, posterior to the sartorius, lying altogether anterior to the bursal abscess which, if extensive, encroaches on the parts behind and below its earlier position.

All my cases show, that in direct contrast to the majority of cases of hip disease, pain was not well-marked. In none was it the reason for the patient seeking advice. The starting night pains frequently noticed by patients, were also conspicuously absent. Further, accepting Howard Marsh's⁴ statement that morbus coxæ most frequently has as its commencement a lesion of the osseous and not the synovial tissues of the joint, we should commonly

² Treves' System of Surgery, vol. ii., p. 37.

Erichsen's Science and Art of Surgery, 1895, vol. ii., p. 419.

⁴ Diseases of Joints and Spine, 1895, p. 385.

expect to cause pain by jarring the limb, by pressure on the great trochanter, or upwards on the limb below ; this does not result in bursal disease. In one of my cases, definite negative evidence is given as to this point in the report.

The attitude of the limb is perhaps that characteristic in which the two diseases are most alike, for, as would naturally be expected, distension of the bursal cavity gives rise to abduction and rotation outwards of the hip-joint, owing to an attempted relaxation of the gluteus maximus. But in none of the cases is flexion marked ; therefore, as each report quoted below notes, there is no lordosis.

This same abducted position of the limb, just as in morbus coxæ, gives rise to apparent shortening, which by careful measurements, both of the whole limb and of Bryant's triangle, as compared with the other side, is easily shown to be entirely due to attitude, and not real.

Lameness, as evidenced by a limp, resulting from the position of the limb, is often seen with chronic bursitis, and is naturally of no value as a diagnostic sign.

But by far the most conclusive evidence in physical signs is obtained by the use of passive movements. For flexion, abduction and rotation outwards—a combination of movements which is generally observed to be extremely painful in actual hip disease—is, in the disease under notice, either entirely painless or only very slightly felt. In all of the cases quoted, which I had myself an opportunity of examining, I can testify as to the value of this negative sign. Further passive movement is usually free through a normal arc, care being taken in making this examination to completely flex the sound limb. This physical sign, again, is brought out in most of my cases, though in some, as Ashby and Wright⁵ point out, movement is free within only a limited range.

It seems extremely curious when the close similarity of these signs to those of hip disease is taken into account, that none of the text-books usually read (Treves' System, 1895 ; Erichsen's Science and Art, 1895 ; Rose and Carless ; Walsham ; Howard Marsh's Diseases of Joints, 1896) give the question more than a

⁵ Diseases of Children.

passing reference, and none of them point out the signs on which the diagnosis must be based.

However, there are numerous records of isolated cases, amongst which I quote the following as having many common characteristics with my own.

Pridgin Teale,⁶ in recording three cases of tuberculous gluteal bursitis, deplored the paucity of the literature on the subject. Two of his cases came under his notice with sinuses, the third for incision of the suppurating bursa. He concludes:—

1. All date from a blow on the great trochanter.
2. No hip disease could be discovered in any of these three, though administration of chloroform was necessary before deciding in one of them.
3. All recovered rapidly after free incision and drainage. Those two in which the gluteus maximus tendon was divided, healed most quickly, and this he considered a great point in treatment, as the moving tendon was liable to keep up the trouble.
4. Possible confusion may exist in diagnosis between a psoas abscess and an enlarged trochanteric bursa.

The publication of these cases called forth from McNab⁷ the report of two very similar ones. In both, the movement of the hip-joint was much diminished, and the first stage of morbus coxæ very closely simulated, but there was no pain on pressure over the head of the femur, nor on sharply striking the heel or knee towards the hip-joint. Incision brought about a perfect cure, with free movement in all directions.

D'Arcy Power⁸ quotes six cases, of which five affected the great trochanter bursa, and he points out the possibility of secondary involvement of the hip-joint, either along the lymphatic tract, or by direct extension.

Mitchell Banks⁹ has had two cases: in the first, difficulties in diagnosis similar to the above were met with, and in the second the enlargement of the bursal sac was considered to be a fatty tumour, and was dissected out.

⁶ *Lancet*, 1870.

⁷ *Lancet*, 1870.

⁸ *Diseases of Children*, p. 140.

⁹ *Liverpool Med. Chir. Journal*, 1882.

An exceedingly valuable paper on this subject was read by Brackett¹⁰ before the American Orthopædic Association, and many of his observations are worthy of quotation. He says, in noticing the similarity to hip disease in the early stages, that as it is claimed that the bursal disease may extend, and later involve the hip-joint, the early condition may be easily lost sight of, and early active treatment of the bursa omitted. He classes the symptoms under three headings:—

1. Irritation of the joint from contiguity of inflammation.
2. Mechanical effect on the muscles.
3. Possible pressure on the sciatic nerve.

At the discussion which followed, a case of psoas abscess pointing over the great trochanter was mentioned, and it is from this disease also that diagnosis of bursal enlargement must be clearly made.

It must be very unusual for a psoas abscess to point in this position, and a careful examination of the spine in the dorso-lumbar region, should exclude the common precursor of psoas abscess, though flexion and rotation outwards of the hip, and limp, will be common to both, and the similarity of these signs demonstrate that not only in morbus coxæ, but in all lesions around the hip-joint, spinal caries and its sequelæ must be carefully and systematically excluded.

From disease of the subjacent epiphysial line of the great trochanter, chronic tuberculous bursitis is much harder to diagnose, and I can only support my opinion as to the causation of my cases by the facts that no carious bone was found at the operation.

In several the rapid healing after incision and scraping, the absence of bone night-pains, and the permanence of cure after the initial operation—all of which are by no means usual in tuberculous caries of bones in other parts of the body—uphold this view.

Coming to the question of prognosis.

As compared with the prognosis in hip disease, in bursal tuberculosis the majority of cases show a rapid recovery, with free movement, a fact which will much enhance the value of a

¹⁰ Trans. Amer. Orth. Assoc., 1887.

correct diagnosis. But it is well to notice that several writers refer to the possibility of a secondary involvement of the hip-joint, though I have not found records of any cases. It seems that Case 5 (A. B., under Mr. Dunn) is a case in point, and for this reason, until a satisfactory period has elapsed in which the joint has remained free of the disease, the prognosis must be guarded.

The treatment in the hands of all the surgeons I have quoted has been on much the same lines, consisting in as free removal as possible of the tuberculous abscess wall, with fixation of the limb in a Bryant's or Thomas's splint until complete healing has taken place.

Beyond the cases quoted by Pridgin Teale, we have no evidence as to the value of division of the gluteus maximus tendon.

The other bursa affected by this disease of which I am able to produce cases, is that situated over the inner surface of the tibia, under the sartorius tendon, around the tendons of the gracilis and semi-tendinosus.

Here the more interesting question of diagnosis is from periosteal abscess of the tibia, and it is not necessary to do more than refer to signs which I have suggested as helpful in the first set of cases when disease of the great trochanter was being considered.

In Mr. Jacobson's case the sartorius tendon was found in the abscess cavity, and divided, which is a very strong point in the diagnosis.

In connection with this case it is further interesting to note that division of this tendon seemed to have little influence on the disease as contrasted with Pridgin Teale's opinion as to the benefit of division of the gluteus maximus alluded to above.

In both the cases (6 and 7) in which this bursa was diseased it was a question of considerable difficulty to decide that the knee-joint was free from disease, and I think the question was certainly decided in the negative with regard to Case 6 (H. P.), and on Case 7 (C. D.'s) first admission his knee-joint was thought to be healthy, but now the extension of suppuration across the limb to the upper and outer side of the joint, and the limitation of movement are extremely suggestive of a secondary involvement of the periarticular and subsynovial tissues.

Below will be found notes of seven cases. Cases 1 to 5 inclusive are instances of tuberculous disease of the bursa over the great trochanter, and cases 6 to 7 of the sartorius bursa. In conclusion I must express my thanks to the members of the Surgical Staff of Guy's Hospital who have permitted me to make use of the following cases; it will be seen that three were under the care of the late Mr. Davies-Colley, who had kindly promised me notes of other cases of this disease, a promise which those of us who recall his accuracy of observation must all regret his inability to carry out.

CASE 1.—T. L., male, æt. 12. Admitted into Luke ward under Mr. Davies-Colley on July 26th, 1897, for a sinus over the right hip.

Early in July, 1893, he received a blow over the right great trochanter, and a fortnight later was admitted into Guy's under Mr. Davies-Colley, when an abscess under the gluteus maximus tendon was incised and scraped.

In February, 1894, the swelling reappeared, and when readmitted in March, there was no signs of the hip-joint being affected. The bursal sac over the great trochanter was dissected out and pronounced tuberculous.

When re-admitted on July 26th, 1897, the "sore" had been present a month. There was no pain. It extended from half an inch below the right great trochanter, for one and a half inches down thigh vertically. In the centre was a spot which discharged clear fluid. There was some reddening of the surrounding skin. Movement of the hip-joint was quite free in all directions, except in extreme circumduction. Jarring the joint caused no pain. Both legs were of the same length, though from slight abduction of the right limb, this appears slightly lengthened. No lordosis. There was no recognisable change in the bone.

Diagnosis.—Tuberculous bursitis over great trochanter.

Under gas and ether the sinus was explored, and no connection with joint or bone found. It was washed out with Lot. Hydr. Perchlor., and filled with Iodoform Emulsion. A double Bryant's splint was applied, and patient was discharged on September 1st, with the sinus almost closed.

I had no opportunity of examining this bursa microscopically.

July 6th 1900. His Mother writes that, "the hip is thoroughly satisfactory. There is no sore or lameness."

CASE 2.—A. T. P., æt. 24, male. Admitted into Luke ward under Mr. Davies-Colley on April 29th, 1898, for a discharging sinus over the left hip.

The swelling first appeared three years ago over the left hip, and burst after sixteen weeks. After discharge had lasted a year he was operated on in the Bucks Infirmary, and is stated to have been cured. In March, 1897, a second swelling appeared in the same place and burst in August, and in January of this year another came below and burst recently. The two sinuses have persisted. He has continued working, though with considerable pain, especially on performing rotatory movements.

On examination, the old scar and two sinuses are found over a somewhat thickened great trochanter. Movements of the hip are free and painless; no lordosis.

Under A.C.E., the abscess cavity was found to extend upwards under the gluteus maximus for four inches, and also downwards over the vastus externus; it was considered to be the bursal cavity and was filled with tuberculous granulations. Several sinuous tracks were opened up, but none were found connected with the bone. The cavity was packed with sulphur on iodoform gauze, partly sewn up, and weight extension applied.

The wound was quite healed on his discharge on July 2nd.

I had no opportunity of making a microscopical examination.

He writes, July 8th, 1900, to say that his hip is well.

CASE 3.—H. H., male, æt. 12. Admitted under Mr. Howse on March 12th, 1898, for a swelling in the thigh.

At six years of age he had bronchitis, and an "abscess in the hand," and from time to time has had various swellings in the neck, which have subsided under treatment.

Two months ago the swelling began without known cause, and painlessly, this gradually increased, the only inconvenience to the patient being a slight limp.

On examination, there is an oval soft fluctuating swelling over the great trochanter, extending downwards over the vastus

externus. The skin over it is colourless. The two limbs are the same length. No wasting of muscles. Temperature varies between 100° and 98°.

Under A.C.E., a four inch incision was made into the swelling, and much pus escaped. The cavity was packed with iodoform emulsion, and was healed on April 18th.

Mr. Howse's diagnosis was a tuberculous bursa over the great trochanter, but no material from the wall was sent for microscopical examination.

In July, 1900, he reports himself quite well but with a slight limp.

CASE 4.—G. L., æt. 27, male. Admitted into Luke ward under Mr. Davies-Colley on February 1st, 1899, for discharging sinuses on the outer side of the right thigh. No tuberculous history.

In August, 1898, a swelling appeared, slightly red, and became as large as a hen's egg, when it burst and discharged pus, and except for one attempt to heal, has since discharged; there was a history of two blows in that region, but neither was immediately related to the appearance of the swelling.

On admission, patient is gaunt and pale; there is a sore one and a half inches long, five inches below the right great trochanter; beneath the sore is a well-defined freely movable mass. The sore discharges pus at two places. Movements of the hip-joint are quite normal. The superficial inguinal glands are enlarged. Under an anæsthetic Mr. Davies-Colley found the sinus to pass upwards towards the great trochanter for three and a half inches deep to the fascia lata; by prolonging the incision, a cavity, lined with gelatinous nodules and thought to be the bursa, was laid open and scraped. No disease of the bone or subjacent joint was detected. The cavity was filled with Iodoform Emulsion and sewn up.

When discharged on February 25th, the wound had quite healed, and the condition was quite satisfactory.

Mr. Davies-Colley's diagnosis here was tuberculous bursitis, and fragments of the abscess wall, removed for examination, showed all the usual appearances of tuberculous granulation tissue, but

the process of caseation was in many places far advanced, and no tubercle bacilli could be found.

This case has relapsed, and now (July 7th, 1900) he has much discharge with some pain.

CASE 5.—A. B., female, æt. 5. Admitted into Martha ward under Mr. Dunn, for a swelling over the right hip, on March 20th, 1899.

In January last, after a fall, the parents noticed a swelling over the right buttock, and that patient also complained of some pain.

On admission, the right leg is slightly inverted. There is no shortening. Flexion is free and complete, but extension is very slightly limited. There is no pain on movement. Rotation outwards is slightly less than on the opposite side. There is an oval swelling over the great trochanter, which can easily be felt, and seems to be entirely normal.

Under A.C.E., Mr. Dunn made an incision over the right great trochanter, and six ounces of clear yellowish fluid escaped. The cavity extended upwards under the gluteus maximus for some distance; the great trochanter was quite normal. Some pieces of the thickened cyst wall were removed for examination, and the cavity was scraped and flushed with boiled water. A continuous suture was used, and a splint applied. The wound healed by granulation, and was completely closed on patient's discharge on May 7th. There was no further pain in the hip.

I reported after microscopical examination:—"On section, the wall of the cyst over the great trochanter consists of granulation tissue, having all the usual appearances of tuberculous tissue. No bursal epithelium can be distinguished. No tubercle bacilli can be seen after staining the sections by the usual method."

On January 22nd, 1900, patient was readmitted with a stiff hip-joint, which is fixed with flexion of some 15°. Very little movement is possible, and this is accompanied by pain. The scar opened a few weeks ago, and has since discharged. A double Bryant's splint was applied, and the sinus dressed antiseptically, under which treatment it healed, and when patient was discharged three weeks later in a splint, movement at the hip had increased and was painless.

Mr. Dunn has kindly given me his opinion that he now suspects secondary invasion of the hip-joint.

CASE 6.—H. P., æt. 13, male. Admitted under Mr. Golding-Bird on June 28th, 1899, for a discharging sinus below the left knee.

A year ago he noticed a swelling over the front and inner side of the internal tuberosity of the left tibia, which caused some pain. This was lanced by a doctor, and pus escaped, since which time discharge has continued, but is now more watery. No bone has ever come away, and patient has always been able to move his knee freely.

On admission, there is a small round raw area three inches below the inner tuberosity of the tibia, exactly over the insertion of the sartorius; the edge of the ulcer seems bound down to the deeper parts and emits a watery discharge. The movements of the knee are quite normal; no pain on any part of the tibia around, and no thickening of the bone.

Under A.C.E., the sinus was enlarged and found on probing to extend in all directions over the inner surface of the tibia. No bare bone was discovered, and the cavity did not extend upwards towards the knee. The cavity was scraped and filled with iodoform emulsion and sewn up. Healing by primary union ensued.

On microscopical examination of the scrapings from the abscess wall it presents the typical appearances of a chronic tuberculous granulating cavity. No tubercle bacilli could be detected.

July, 1900. There is no pain, swelling or lameness.

CASE 7.—C. D., male, æt. 20. Admitted on January 4th, 1899, under Mr. Jacobson, for a discharging sinus on the inner side of upper end of the right tibia, and a swelling beneath it.

He first noticed the swelling a year ago, when it was painted with iodine, and improved so that he could get about, but with occasional twinges of pain. After six months it began to swell again, and, the skin becoming inflamed, was opened by Dr. Pritchett, of Rochester. Discharge has continued until his admission. The right leg is swollen, especially on the inner side of the tibia, just below the knee. Here there is a sinus with continual discharge. There is no pain, even on walking. Patient

can flex and extend the knee quite freely and to the full extent. The knee-joint itself contains no fluid, and does not appear to be affected. There are enlarged inguinal glands.

On January 6th, it is noted, the swelling is about the size of the palm of a small adult hand, uniform, doughy, and somewhat pulpy to the feel. The actual synovial outline of the joint does not appear to be involved. Movements of the joint are free and smooth, and there is no grating. Over the front of the swelling is an ulcer the size of a sixpence, covered by blueish-red granulations.

Under an anæsthetic the sinus was laid open by a crucial incision, and a quantity of caseous material was removed. Pouches passing forwards and backwards, were opened up, and another passed under the tendon of the sartorius, which was exposed in the cavity. The tendon was divided to expose the cavity completely. The cavity was freely scraped, and iodoform emulsion poured in. No sutures were used.

A note states:—"This is probably tuberculous disease of bursa beneath the sartorius tendon. This is made probable by the absence of bone disease. The joint itself is not involved by the sinus leading underneath the tendon of the sartorius."

The discharge had been examined by me before operation for evidence of sarcoma, but none was found. No scrapings from the operation were obtainable for examination.

January 21st. The granulating area was grafted by Thiersch's method; all the grafts adhered, and the wound was soon nearly healed.

He was discharged on February 25th with the area a firm cicatrix; movement after so much fixation is good, but extreme flexion and extension are impossible; there is no pain.

On April 26th, 1899, he was re-admitted with two sinuses in the old scar, which is a large depressed area; these were scraped. An exploring syringe was introduced into the knee, which seemed swollen; no synovial fluid was withdrawn. The wounds had healed by May 26th, when he was discharged.

In connection with this case the more recent history is of interest, for he was re-admitted under Mr. Jacobson in January of

this year for a swelling above and outside the right knee; much pus was found on incision, but no connection with the knee-joint or the femur could be made out, though such was considered quite likely to be present. It is noted that movement of the knee-joint is limited, flexion being only possible to a right angle, and extension limited at 15° from the straight position. Later on a further tuberculous focus was scraped out in the position of the old bursal sac by Mr. Steward, who also, when patient was last admitted in May of this year, scraped two sinuses which had persisted. Movement remains limited as on the last admission, and involvement of the knee-joint is strongly suspected by Mr. Steward.

ON THE TREATMENT OF CHRONIC MIDDLE EAR SUPPURATION BY REMOVAL OF THE MALLEUS AND INCUS.

By C. H. FAGGE, M.S.

A LARGE proportion of the cases attending any aural department are those of chronic middle ear suppuration, and I think this large proportion becomes a majority when the class of patient attending is of the poorest. Owing to its extreme frequency, it cannot be wondered at that so little importance is attached to it by the patients themselves, and that grave results, such as facial paralysis, mastoid abscess, with intracranial complications often occur. There are few subjects upon the danger of which there is more need to warn patients than chronic middle ear suppuration, and it is even more to be regretted as showing the slow progress that education in these matters has made, that such neglected dangers are scarcely less common in even well-educated and common-sense people, who speedily pay attention to any other slighter but certainly far less dangerous lesion. Perhaps the apathy of the public towards this constant danger is in part due to the belief, which certainly was correct till recent years, that many cases of this kind could not be cured, or at least the probability of cure was not so great as to repay much trouble in their treatment.

Up to the early eighties, the chief, if not the only method at our command, was antiseptic syringing, or insufflation, the commonest drug used being boracic acid. Therefore, before

beginning a discussion of those cases which do not get well by non-operative methods (which cases are the subject of this paper), it will be best to review the more recent methods of treatment.

Firstly, I can only add to that of many others, my small experience of the immense importance, when one is satisfied that suppuration is coming from the middle ear, of an examination (with mirror or finger) of the nasopharynx. Many cases could be easily produced to prove this, and yet it is surprising how often this is neglected, even when otorrhœa is associated with such symptoms as indistinct speech, mouth-breathing, and frequent colds, which in themselves must provoke a strong suspicion of the presence of post-nasal growths. One is constantly struck with the large amount of improvement in middle ear suppuration resulting from the removal of an amount of adenoids even so small as to escape the detection of an imperfectly trained finger, and the results of removal of these growths, when in large amount are everywhere obvious. It is well worth remembering that soft vascular adenoids, and not the dense fibrous variety, are often associated with middle ear suppuration, and as these do not till present in very large amount cause nasal obstruction, obvious symptoms of their presence are usually absent. Therefore, examination of the nasopharynx must be a routine procedure in all cases of middle ear suppuration.

Secondly, it will serve my purpose briefly to point out that there are now at our command several better antiseptics than boracic acid, for the following, of which I have, under Mr. Laidlaw Purves, made extensive use, are some of many now generally accepted as valuable :—

Lotio Formaldehydi	...	G.H.P. 1 in 500—1 in 250
Lotio Lysol	1 per cent.
Lotio Chinosol	1 in 400
Lotio Acid Carbol	1 in 60—1 in 20
Guttæ Spir. Vin. Rect.	33 per cent.

I give the usual strengths employed, as knowledge of the suitability of some of these drugs for the ear does not seem widespread. In my experience formalin has given perhaps the best results; but lysol is very effective in cases of much discharge

with profuse epithelial desquamation. It has a good power of penetration, and serves the double use of syringing with an alkaline saline solution, followed by antiseptic drops, which is the common practice of some aurists.

Thirdly, application of chromic acid to granulations, curetting of granulations, and removal of polypi, all have their place, and perhaps I may be excused for insisting on the necessity of all possible antiseptic precautions before the two last-named modes of treatment are adopted. The removal of a polypus from a meatus filled with virulently infective discharge leaving its torn base as an excellent spot of inoculation for a deeper, and often dangerous, if not fatal, infection, is directly opposed to all present bacteriological teaching; but before this scientific aspect of the question was recognized, Macewen,¹ in the larger field of antral operations, has pointed out that granulations are often protective where the possibly exposed dura mater for a long time maintains its immunity when covered with granulations, and their removal may give rise to re-infection. In fact, there are few hints of treatment which have been more generally useful in otology than a liberal interpretation of Listerism, which has long been insisted on by Urban Pritchard. A "moderate asepsis" (if the term is allowable) is easily obtained, and then the curetting or removal of a polypus acts only for good.

Fourthly, another line of treatment, no doubt the outcome of the above, which, too, as far as I am aware, we owe to Urban Pritchard and Arthur Cheate, is antiseptic packing, which is, briefly, thorough purification of the external auricle, meatus, &c., consecutively with soap and water, carbolic, or other antiseptic lotion, and instillation of 1 in 20 carbolic acid, or Lister's strong mixture for ten minutes, and packing with sterilized cyanide gauze, to be changed every one, two, or more days, as is deemed necessary.

The above are generalizations on the usual lines of treatment of chronic middle ear suppuration, and are merely quoted as examples, though other remedies, possibly even more suitable for individual cases, readily occur to one's mind. Yet, as all will agree, these frequently fail, even when conscientiously and per-

¹ Macewen: *Pyogenic Disease of Brain and Spinal Cord*, p. 74.

sistently used over periods of months, to do more than keep the discharge in check, while if treatment is discontinued for even a short period, it resumes its former volume, and a discharge, even if checked in amount, is still a constant danger. It would no doubt be of much interest to produce statistical evidence as to the proportionate failure of six months' careful antiseptic treatment, and to analyse the cases ; but methodical treatment in the very poor is rarely carried out, and, again, the cases which come to a hospital are always the more serious ones, which elementary treatment has failed to cure ; so a statistical enquiry would certainly be open to many fallacies. The following, however, will serve as examples :—

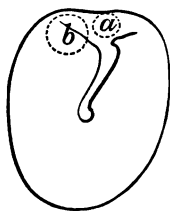
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|-------------------------|---|
| 1. Case 9. E. M. | } <i>Vide</i> the notes of the cases below. |
| 2. Case 14. P. H. H. P. | |

In others of my cases, though prolonged treatment was ordered before operation, it is difficult to prove that it was carried out, but in the above no doubt exists on this point.

Why does such treatment fail ?

Perhaps this question can be most profitably answered by detailing the common characteristics noticed on inspection of all the cases, and then discussing their anatomical and pathological interpretation. A curious fact is now revealed that in all the cases dealt with by operation in this paper, there existed one of two varieties of perforation usually surrounded or blocked with granulations.

- (a) In Shrapnell's membrane, immediately above short process of malleus.
- (b) In the posterior superior segment just beneath, or involving the posterior malleal fold.



Right Membrana Tympani, giving a diagrammatic representation of perforations *a* and *b*.

These two perforations are generally agreed to indicate suppuration in the attic (*recessus epitympanicus*), which, it is scarcely necessary to mention, is the upper compartment of the middle ear, in which lie the head and neck of the malleus, and the body of the incus, and which superiorly is separated from the meninges and the temporo-sphenoidal lobe by the very thin plate of the petrous, known as the *tegmen tympani*.

Attic suppuration accounts for quite a fair proportion of all cases of purulent middle ear disease. In an examination of 375 cases, Milligan² found the attic affected in 12, 3·2 per cent., and Randall found 65 in 2,500, or 2·6 per cent. Judging by the Guy's out-patient department, I should have put the proportion much higher; but I have no statistical evidence, and again, as I pointed out, these are the chronic cases, and therefore likely to seek advice. All writers emphasize the chronicity of disease in this region, and draw their reasons for it from anatomical facts, which show that this portion of the middle ear is, by folds of the mucosa, completely shut off from the lower two-thirds of the cavity. It is further subdivided by septa of mucous membrane, passing along the ligaments of the ossicles and the tendon of the tensor tympani, into smaller compartments, in which suppuration which has ceased in the main cavity would naturally be most prone to persist. Again, the space is most inaccessible to treatment, as only the lower part of the outer wall is in the natural condition bounded by the *membrana tympani*, and therefore alone can be reached from the meatus. Even Hartmann's ingenious attic syringe has been of little use in cleansing the small cavities of the attic, and all are agreed that treatment of attic suppuration is extremely tedious. All my cases treated by operation show this; and, further, the following are examples from Mr. Purves' out-patients of some twelve cases which I have had under personal observation for some months, and which have up to the present completely exhausted the simpler modes of treatment known to me. Most of them are awaiting operative interference.

1. H. L., æt. 10, came to Mr. Purves' out-patients on January 2nd, 1900, with 12 months' Rt. otorrhœa. He has a large

² *Lancet*, 1892, vol. i., p. 136.

posterior perforation involving the posterior malleal fold, which exposes the head of the stapes and shows a vacancy which should be occupied by the descending process of the incus. Conscientious syringing with formalin (1 in 500) has not until now, May 22nd, done more than slightly diminish the discharge. The perforation shows no signs of healing.

2. K. D., æt. 24. July 4th, 1899. About three years ago she noticed a discharge from the right ear, which soon ceased. A year ago it began again, and has since continued. On examination, there is a small, moist perforation guarded by granulations in the middle of Shrapnell's membrane, just above the short process of the malleus. She has used carbolic lotion, chinosol, spir. vin. rect., and formalin without appreciable benefit, and has also had the attic syringed out with Hartmann's canula on several occasions. Now, on May 22nd, 1900, the perforation is much as when she first came, but because patient was averse to operation, and also as a test case for the purposes of this paper, I have delayed more radical procedures.

3. F. H., æt 17. January 30th, 1900. Has had deafness, with intermittent discharge from the right ear, since measles, ten years ago. He has a typical (*b*) perforation. Hearing $\frac{R. \frac{1}{2} \text{ in.}}{L. 36 \text{ in.}}$ (normal). He has used formalin and lysol ever since January without appreciable benefit.

The anatomical relations of these perforations are very suggestive:—

(*a*) To the head of the malleus and body of incus.

(*b*) To the descending process of incus.

And it has been shown, and now conclusively proved, that the perforations in these positions indicate usually disease of the malleus or the incus.³

The perforation (*a*) is perhaps the commoner, and certainly the more strongly indicative either of caries of the head of the malleus, body of the incus, or both, caries of the attic wall, or of collections of cholesteatoma in the attic.⁴

³ Grunert, *Archives für Ohrenheilkunde*. Vol. xxxiii., p. 207.

⁴ A. H. Cheatele, *Operative interference on the drum and ossicles in chronic Middle Ear Suppuration*. *Practitioner*, October, 1898, p. 362.

A typical (*b*) perforation is uncommon ; the process of destruction of the membrane often advancing so that the well-known horseshoe perforation is produced, or a posterior perforation showing the head of the stapes, and which by showing the position to be vacant, which should be occupied by the descending process of the incus, suggests its own causation. Chéatle (*loc. cit.*) originates an observation that from a typical (*b*) perforation the suppurative process may extend upwards, or involve the attic secondarily.

A further confirmatory point in the examination is the occasional detection of carious spots of bone with a Hartmann's probe.

We have now a ready answer to our question as to the cause of failure of treatment in these cases :—

1. Caries of bone.
2. Defective drainage from the middle ear by a superior perforation.

Before going further, it must be undoubtedly admitted that even with such lesions present, healing does take place occasionally, and on this ground Politzer⁵ opposes operative treatment in the slighter cases, pointing out that "circumscribed caries on the hammer and incus may heal, and that the head of the hammer and the whole of the incus during long-continued middle ear suppuration, may completely disappear through carious degeneration."

Gompertz⁶ considers that a cure of circumscribed caries of the malleus and incus is possible by tympanic irrigation ; while Milligan supports Schubert in his opinion that attic irrigation is unsatisfactory and usually futile.

The following cases, under Mr. Purves, illustrate the process of natural healing of an "attic" perforation :—

4. A. M. T., æt. 32. April 24th, 1900. Has had pain in and discharge from left ear for ten months, the discharge ceasing two months ago. The pain, however, is worse, and deafness is progressive. She has a large posterior perforation, now covered with a thin cicatrix, which exposes the head of the stapes ; no

⁵ Diseases of Ear, 1894.

⁶ Monatschr. für Ohrenheilk, 1892-1893.

signs of the descending process of the incus to be seen. She has very defective hearing; watch left ear 4/100"; pain and tinnitus intermittent; patient has since much improved by periodical use of Politzer's bag.

5. E. C., æt. 54. March 27th, 1900. Attended here for discharge twenty years ago, "when something (? polypus) was removed from her right ear." She has attended at intervals of three or four years ever since for the same trouble, and is now becoming more and more deaf. On the left side the outer attic wall is eroded; Shrapnell's membrane is replaced by a thin cicatrix, which exposes the head of the malleus; the cicatrization also extends forwards, involving the anterior fold. On the right side the condition is exactly similar, but the area of cicatrization is smaller. Hearing by meatus:—Watch $\frac{\text{R. contact}}{\text{L. nil.}}$ Rinne's test negative.

6. W. S., æt. 39. April 3rd, 1900. Patient had discharge from the right ear when about two years of age; this continued for 27 years, and then ceased without any treatment worth mentioning. His present trouble is extreme deafness, tinnitus and giddiness, which comes on in attacks; much of his deafness is no doubt due to an internal ear lesion as Rinne's test is positive, and there is much loss to the fork on the mastoid process. The right membrane is entirely destroyed, and Hartmann's probe passes easily into the attic, the outer wall of which has remained; the malleus as far as can be seen is absent, but the lower part of the body and descending process of the incus and the incudo-stapedial joint are easily seen. Radiating from the head of the stapes to the inner tympanic wall are a large number of very firm adhesions.

7. F. C., æt. 29. March 13th, 1900. Has a purulent discharge from left nostril, and suffers from frequent attacks of sore throat. She had a continued discharge from right ear "some years ago." On examination, there is pus in the right side of nose, no polypi, and no other evidence of antral empyema; much pus with post-nasal growths seen in nasopharynx. She has a healed perforation, involving the posterior malleal fold in the right membrane; this cicatrix exposes the descending process of the incus articulating

with the head of the stapes: the incus, as far as can be seen, is complete. Watch (normal 36 ins). $\frac{\text{Rt. } 1}{\text{L. } 1}$ improved by Politzerisation.

With two others, the notes of which are not available, these four cases are all that I have come across in the last eighteen months at Guy's, which show a natural healing of what is known as an "attic" perforation, and of these, perhaps the suppuration in No. 1 has ceased too recently to be pronounced a cure—for there is nothing more characteristic of such cases than the intermittence of discharge for short periods (Case 15 exemplifies this excellently). Again, in support of my argument it may be urged that in Cases 2 and 4 there is no definite evidence of ossicular caries ever having existed. Most writers on this subject ignore this point of the natural healing of "attic perforations," but Milligan⁷ can recall a few cases of cure of perforations in Shrapnell's membrane by simple methods: he thinks that these have existed but a short time, and in none of them was caries of the ossicles demonstrable.

All these three cases are interesting in the extremity of deafness and the severity of the subjective symptoms which have accompanied the process of natural cure, and these symptoms in themselves justify recourse to a more active line of treatment.

Therefore, with all deference to such a master's opinion as Politzer's, it is now generally agreed to be inadvisable, when the small amount of destruction by caries of the ossicles which takes place in ten years is considered, to expose the patient to an almost life-long risk of infective complications, so that the ossicles may be removed by the law of nature rather than by a surgeon. It is, however, interesting to note, as a guide to operative interference, the method of the natural cure of such suppuration, and it may be briefly stated to be in two directions:—

1. Erosion of the outer attic wall.
2. Total caries of the ossicles affected, and more or less destruction of the membrane.

Both bring about a good result by ensuring free drainage, and the latter also by removing the usual cause of the suppuration.

⁷ Brit. Med. Journ., 1893, vol. ii., p. 565.

Above it has been pointed out that certain forms of perforation are considered to indicate disease of the malleus, incus, or walls of the attic, and it will be profitable now to discuss the relative frequency with which these different bones are affected. In this connection, a fact often lost sight of by those who consider that caries of the ossicles is infrequent, must be mentioned, namely, that the mucous lining of the middle ear acts as a nutritive periosteum to these bones. Probably caries of the ossicles is rarely, if ever, a primary local infection, but usually spreads to the bone from destruction by ulceration of the superjacent lining membrane of the tympanum; infection of the lining membrane itself is nearly always nasopharyngeal in origin by extension along the Eustachian tube, and never a sequel of otitis externa suppurativa; possibly in some small proportion of cases it may be a metastatic infection by organisms carried through the bloodstream, but this must be a pure surmise. The bacteriological aspect of carious processes in the middle ear has not been worked out apart from the bacteriology of chronic middle ear suppurations in general; the chronicity and general characteristics of the process suggest that it may be more often than has been appreciated of a tuberculous nature, and possibly in the future we shall find that the prognosis of these cases must be based on a bacteriological examination rather than on the microscopical appearances of the lesion in the membrana tympani; the uniform presence of pyogenic cocci cannot be taken as truly causative. Dench,⁸ recording twenty-four cases of ossiculectomy for chronic suppurative otitis, found the incus carious or destroyed in all. Grunert (*loc. cit.*) found the same bone diseased in twenty-five out of twenty-eight cases; Ludwig⁹ in sixty-four out of seventy-five, and with this consensus of opinion, Milligan¹⁰ agrees that the incus is the bone most often diseased. Panse (German Otolog. Congr. Dresden, 1897) gives as the signs of isolated caries of the incus, the following:—

1. Chronic scanty watery discharge.
2. Perforation in posterior superior quadrant.

⁸ N. Y. Eyes and Ear Infirmary Reports, 1893, p. 44.

⁹ Archiv. fur Ohrenheilkunde Bd. xxx., p. 24.

¹⁰ Lancet, 1892, vol. i., p. 136.

3. Frequent preservation of the limbus in that place.

4. Polypi or pus from above.

Ludwig found both malleus and incus affected in 50 per cent. of his cases, a condition which existed in twenty-one out of twenty-four of Dench's cases.

Schwartz¹¹ is of opinion that caries of the stapes is very uncommon. Dench found this bone diseased in two of his twenty-four cases. This immunity of the stapes is no doubt due to its double blood-supply.

It therefore appears that persistent chronic suppuration can in a large proportion of cases be demonstrated to be associated with, and I think it will be granted is due to caries of the malleus and incus; and this condition prompted Schwartz¹² to remove these two outer ossicles, excising at the same time the remains of the membrana tympani. But before this procedure can be looked upon as a certain cure of the otorrhœa, it must be shown that the carious processes have not extended to the parietal tympanic wall, and as far as we can at present judge, this complication of chronic suppurative otitis media seems to be far less frequent than caries of the small bones, which is not to be wondered at when the isolated condition of the latter, easily cut off from their vascular supply by inflammatory processes, is taken into consideration. Dench found a carious attic wall in seven of his twenty-four cases; Milligan thinks the parietes are often affected.

The judicious use of Hartmann's probe, both before and at the time of operation, may help to determine this point, and in every case where it is suspected, justifies curetting of the eroded bone, but its frequency can only probably be correctly estimated by a careful examination of those cases in which ossiculectomy has failed. These can afterwards only be satisfactorily treated by antrectomy, though it is well worth while to wait for a time after removal of the ossicles and a thorough curetting of the carious bone, for no doubt this may sometimes prove effective, as in the case of M. P. (Case 13 below).

¹¹ Die Chir. Krankheiten des Ohres, 1884.

¹² *Loc. cit.* page 389.

It will now be granted, I think, that after failure of simple methods, with the indications just detailed, the ossicles must be removed. I do not propose to go into any operative detail, but merely to point out that antiseptic precautions must be extremely carefully carried out. I have tried the operation under local anæsthesia and failed (10 per cent. cocaine being used); nearly all operators now agree that a general anæsthetic is necessary, and the meatus can then be dilated if necessary with dressing forceps to admit the largest sized Pritchard's speculum. Removal of the incus is by far the most difficult part of the whole process, and no further evidence of this is necessary than to point out the many and varied instruments which have been devised for this purpose; again, I have found it very difficult to decide in cases where disease of the incus was diagnosed as far advanced (by the perforation) and a fragment only of the incus could be removed, whether the remainder of the incus had been destroyed, or in process of removal had broken off and remained in situ, where it would be a very potent factor in continuing the trouble. After excision of the membrane and extraction of the malleus and incus—the tympanum must be syringed out with 1 in 20 carbolic acid solution, and then packed with a strip of boiled gauze. This is usually removed on the fourth, eighth and fifteenth days, when the cavity has usually healed. Reinhardt,¹⁸ in a record of twenty-three cases, notes that cicatrization took place in from eight to twenty-one days. Dench (*loc. cit.*) says the time varies greatly, during which the granulations are being covered with epidermis—being usually from three to six weeks.

The results must be studied from two points of view :—

1st, as regards curing the discharge.

2nd, as regards hearing.

My own cases are too few to draw any valuable conclusions with regard to either question. The following, however, is valuable evidence from the hands of other operators.

First, with regard to cessation of discharge.

¹⁸ Archiv. f. Ohrenheilk. v. 33, p. 94.

Reinhardt (*loc. cit.*) had fifteen recoveries in twenty-three operations, removing the malleus in all and the incus in sixteen cases. Dench, in twenty-four cases, by removing the malleus six times, malleus and incus eleven times, and all three ossicles three times, cured the discharge fourteen times, and reduced it in eight. In Milligan's fifteen cases, eleven were cured of the discharge, and in two others it was reduced. Ludwig records (*loc. cit.*) forty-two cures from seventy-five cases, and twenty-eight of these successes were examined by Grunert¹⁴ one and a quarter to one and three-quarter years later, when twenty-two of them were found to be entirely satisfactory.

It is most unwise to press the value of statistics, for while it might be argued that this collection of cases demonstrates the cure of chronic middle ear suppuration in 60 per cent. of cases by removal of the ossicles, we have no details of the duration of otorrhœa, previous treatment, and position of the perforation, although the evidence as to the presence of ossicular disease in some of these (Dench's and Grunert's) cases has been already quoted.

It is clear that in a certain proportion of cases—usually those in which ossicular caries is accompanied by disease of the tympanic walls—this operation of removal of the malleus and incus fails, though it is probable that localised caries of the parietal walls may be cured by ossiculectomy and curetting, excellent drainage being also procured by this means. This happened in two of my cases in which there was certainly caries of the attic wall. Should ossiculectomy fail—discharge persisting eight weeks after the operation—the radical Schwartz-Stacke's operation must be undertaken without hesitation. However, it cannot be too clearly insisted that for this class of case ossiculectomy must be the first operative procedure undertaken, and only when this has failed should the radical operation be proposed. This is not the place to go into a discussion of the relative value of these methods, but all aural surgeons with an experience of both operations will agree that ossiculectomy, by

¹⁴ Brit. Med. Journ., 1893.

being less serious, by allowing the patient to return to work earlier, and by restoring the hearing to a usually greater extent, has advantages over antrectomy which would be, as the records above show, an entirely unnecessary proceeding in these cases of localised attic suppuration.

With regard to the hearing, the evidence is less complete and not so brilliant, and it has been my custom to make no promises of great benefit to patients before operating.

All surgeons with a knowledge of the possible complications of middle ear suppuration will agree that the cure of the discharge is the chief result to be aimed at. As Milligan¹⁵ so happily puts the case, "The result quoad vitam is more important than the result quoad functionem." This writer records benefit of hearing in eight out of thirteen cases—in three it was unchanged, and in two made worse. In Dench's cases, in fourteen hearing was improved, in nine unaltered, and in one reduced. In my own nine cases the hearing was improved or unaltered in all.

Other minor yet important subjective symptoms, as frequent earache, vertigo, and tinnitus, are, as far as my experience goes, always benefited by the operation—the effect on those cases in which these were present will be found detailed in the notes of the cases at the end of the paper.

The dangers of the operation seem to be very few, and in this, among other respects, compare favourably with the more radical Schwartze-Stacke. No fatal case is recorded. In Milligan's first case there was defective taste on the same side of the tongue for some days, probably in most cases the chorda tympani is divided, yet in those of mine, which I had the opportunity of examining, I was unable to find any defect of taste. Two cases have been recorded of a transient paresis of the facial nerve—in the eighth of my operations the anæsthetist noticed one twitch of the facial muscles during extraction of the incus, but there was no subsequent ill result. Dench¹⁶ wounded the jugular bulb in curetting the tympanic floor; septic thrombosis which supervened was relieved by ligature of the internal jugular vein, and the patient

¹⁵ Brit. Med. Journ., 1893.

¹⁶ Journal of Otology, vol. xxvii., p. 297.

recovered. Headache and vertigo commonly occur for a day or two after operation, but are merely transient and easily relieved by morphia, rest in bed, and ice to the forehead. The reformation after a few weeks of the membrana tympani is a result which may no doubt interfere with the marked benefit to hearing which results immediately from the operation. This occurred in seventeen of fifty-one cases published by Lucae and has been recorded by other writers on the subject; it has not happened in any of mine. Should it occur, the newly-formed cicatricial membrane must be excised freely.

Below are details of my nine operations on eight patients (operations 4 and 5 being on the two ears of one patient). Cases 8, 9, 10, 11, 13, and 15, were out-patients at Guy's under Mr. Laidlaw Purves, to whom I am much indebted for permission to operate and also to make use of these notes. Cases 12 and 14 occurred in my own practice. Seven of the above operations were performed in the out-patient department under A. C. E., and that the after-effects were not very serious is, I think, evidenced by the fact that one patient (a woman) was operated on a second time at her own request.

I have purposely made the notes as brief as possible, leaving out all details of examination not essential; but after what I have said about the importance of routine examination of the nasopharynx and nose, it should perhaps be noted that in none of the cases was there any lesion discovered in either of these two cavities which could be regarded as a cause of the middle ear suppuration.

8.—R. C., female, æt. 21. February 14th, 1899. Had been deaf with discharge from the right ear for three years. The trouble came on with an attack of earache; the ear had been operated upon at another hospital and polypi had also been removed when she was an in-patient in Guy's. She had occasional giddiness, and discharge had continued without intermission since its commencement. The right membrane is covered with pink granulations; the left shows a dense cicatrix in both lower segments. There is much foul-smelling pus in the right meatus.

Hearing watch (50in.) $\frac{R. 1 \text{ in.}}{L. \text{ nil.}}$ Rinne, negative both. Fork on mastoid, both normal.

The right ear was syringed with Boracic Acid, Zinc Sulphate and Carbolic Acid lotions, and on the 18th April the right membrane showed a typical perforation in Shrapnell's membrane. —Hartmann's probe was passed into the attic, but could not detect carious bone—the discharge was less foul, but little altered in amount by treatment. Syringing was continued until June 23rd, when the malleus and remains of the incus were removed under A.C.E. Lake's incus hook failed to find any evidence of the rest of the incus in the attic. The head of the malleus showed a large area of erosion on its anterior aspect. There was some discharge on removing the cyanide gauze packing on the eighth day, but this had almost ceased a month after the operation under Carbolic lotion and Guttæ Spir. Vin. Rect., when patient ceased attending without my permission. By the kindness of Dr. Morgan Hearnden, who had sent her to Mr. Purves, I saw patient in April of this year (1900). She had had no treatment since July, 1899, and the otorrhœa had increased.

9. E. M., æt. 18. April 4th, 1899. Scarlet fever eight years ago resulted in deafness and discharge from the left ear. She attended the Throat Hospital, Golden Square, last year, for pain in the head. Her ear was syringed and something removed from it. She now has much foul discharge from the left ear, with tinnitus. The left ear is filled with pus and the whole membrane is granulating. The meatus was cleansed by syringing and instillation of 1 in 20 carbolic acid, and packed with cyanide gauze wrung out in 1 in 20 carbolic; this mode of treatment was continued for three weeks; the otorrhœa continued. Patient, who had been compelled to leave domestic service because of the offensive character of the otorrhœa, was now a ward-maid in Guy's Hospital, as she considered the closer observation there gave her a better chance of recovery. After this her ear was syringed frequently with boracic and carbolic acid lotions and Boracic Acid with Spir. Vin. Rect. instilled with persistence and care. Her hearing (watch 36in) was on the $\frac{R. 4 \text{ in.}}{L. \text{ nil.}}$ and not improved by

Politzer's bag. The fork showed no affection of the internal ear. On June 13th, 1899, the left malleus was extracted with the remainder of the membrane, under A.C.E. The handle of the malleus was much shortened by disease, and the head showed a large area of caries; no incus could be extracted.

The meatus was packed until fourteen days, when the gauze was dry; after this her left ear was syringed at intervals of a week, the alcohol and boracic acid drops being used. Patient's hearing had now improved, and varied from contact to lin. with a 36in. watch. Whispered conversation could be heard at about four yards. May 22nd, 1900, the discharge still remains absent, and tinnitus, which was most annoying previous to operation, has been entirely cured. Hearing with a 36in. watch remains at lin. I think this result may be regarded as permanent.

10. J. P., male, æt. 23. Attended on June 6th, 1899, for pain in head from gathering in the ears. Has had discharge from both since childhood. No history of specific fevers; has had occasional attacks of giddiness. Hearing (Watch 100 in.)
R. 2 in.
L. 5 in. Fork tests show no change in the perceptive apparatus. The left membrane is largely destroyed by a horse-shoe shaped perforation which extends well up into the posterior superior quadrant, involving the posterior fold. On the right side is a smaller anterior inferior perforation. Patient was ordered Chinosol 1 in 1,000 for syringing both ears, and the hearing distinctly improved on the right side, but the discharge continued in both, so on August 15th I removed the left malleus, which was apparently healthy. A few granules were the only remains of the incus which could be discovered. On the 22nd the packing was covered with pus, and Lotio. Ac. Carbol. 1 in 40 was ordered.

On August 29th patient complains of some discharge from the ear operated on since last visit, but no pus is to be seen or mopped out; probe passes easily into the attic, which is quite clear. Watch (100 in.) R. 8½ in.
L. 6½ in. (a gain of 1½ in.). The discharge from the right ear had distinctly diminished, was not offensive, and hearing had improved. After this the lotion was continued

for a short time, and no further note is made. Patient ceased attending without permission.

11. (Operation 4 and 5). B. R., female, æt. 28. March 28th, 1899. Has been deaf in both ears since childhood, dating from otorrhœa after scarlet fever—the present exacerbation of discharge began twelve months ago. She has general headache, tinnitus, and occasional giddiness. On examination, each meatus is blocked with polypi and foul-smelling pus. Hearing by meatus to 50in. watch, is nil; tuning-fork tests show the internal ear to be normal.

Polypi were removed from both ears, and Chinosol 1 in 1,000 ordered for syringing: this was continued until August, when her condition was as follows:—The otorrhœa is abundant from both ears, tinnitus persists, the left membrane shows a posterior superior perforation involving the posterior half of Shrapnell's membrane, and exposing the vacant position which should be occupied by the descending process of the incus; the head of the stapes can be seen deeply in the cavity. August 15th. Ossiculectomy performed on the left ear—the malleus alone extracted; no incus could be found with Lake's incus hook; the extracted malleus showed but little disease. August 29th. No pus in the left ear; noises absent. October 30th. The left middle ear has now granulated over; there has been no discharge from it since operation. Patient says her hearing "is much improved, but it was always her best ear"; unfortunately no data were taken just before the operation, but she hears conversation now for the first time with ease, and the watch at two inches. She is still using Guttæ Sp. Vin. Rect. 33 %, night and morning for this ear.

The condition being so satisfactory, patient desired a similar operation to be performed on the right side. The right membrane showed a large horse-shoe perforation, the handle of the malleus, hanging quite free. Patient had since her first visit used formalin and carbolic acid lotions persistently without reducing the discharge on this side.

October 5th. The remains of the right membrane and the malleus which was healthy were removed; no incus could be

extracted. November 28th. Hearing watch (36 in.) $\frac{R. 4 \text{ in.}}{L. \text{ Contact}}$
 No discharge from either. January 16th, 1900. The left ear discharges; some granulations are to be seen in the floor of the tympanum; covered with pus. The right ear is quite dry; tinnitus and giddiness are absent. Watch (86 in.) $\frac{R. 1 \text{ in.}}{L. 2 \text{ in.}}$

May, 1900. Condition as above; discharge from left ear was now very slight and occasional; right ear still remains dry.

12. A. A. C., male, æt. 25. Sent to me on August 29th, 1899. He had earache when two years old, followed by right otorrhœa, which continued and was uninfluenced by boracic acid powder which was insufflated for some time in 1882; has never had any pain or discharge from the left ear. He has syringed the right ear occasionally, and for the last two months has syringed it regularly with boracic acid on his doctor's advice. Patient now wishes to ensure his life and marry.

Left meatus and membrane normal; right membrane shows a large perforation in Shrapnell's membrane, exposing the neck of the malleus—the opening into the attic is further increased by some erosion of the outer attic wall—the perforation is covered with pus, which soon returns when mopped away. Hartmann's attic probe passed into attic failed to discover any carious spots. Hearing (36 inch watch) right 3; left 20. No nerve loss to the tuning fork.

Ossiculectomy was proposed and accepted, and patient was ordered to syringe the right ear with Lotio Ac. Carbol. 1 in 40, as a preliminary. September 12th, 1899, right malleus and membrane extracted under ether—much bare bone curetted on inner attic wall. Temperature slightly raised for a few days.

October 9th, 1899. Patient was discharged from Bright ward after a week's stay, and the packing removed as the discharge continued, but under the use of Guttæ Spir. Vin. Rect. the latter has much diminished. The whole cavity is granulating well.

December 6th, 1899. Discharge is now occasionally only. Some pus is seen lying in the ear. The radical mastoid operation was now advised, but was delayed for a time at patient's

wish. The neck of the malleus was carious and was fractured in extraction.

June, 1900. Patient writes that he considers himself cured of the discharge, but the hearing has not returned to the normal. His proposal of life insurance has been accepted.

13. M. P., æt. 25. June 27th, 1899. Comes for deafness, discharge and buzzing sounds in the left ear; has continued since childhood with intermission; bleeds occasionally; she was operated on for the bleeding ten years ago; the present attack of otorrhœa began six months ago. Hearing (watch 100 inch). Right 60; left 0. Right membrane normal. Left meatus blocked by a polypus, which was snared.

August 1st. Patient has been using lotio Ac. Carbol 1 in 60 for syringing for the last six weeks; she complains much of giddiness on syringing, and wishes for more radical treatment. There is a small sessile granulation projecting from Shrapnell's membrane; discharge escapes in drops from this region.

September 26th. Three months' treatment having proved ineffectual, the left membrane was incised round its margins and removed. Fragments of malleus and incus were curetted out, but no set ossicectomy was possible, as the bones crumbled away in the forceps. A small carious focus in the attic was curetted.

October 23rd. Discharge has now ceased.

November 14th. Hartmann's probe passes into the attic, which is quite clean and dry. The whole of the tympanum is now covered with epithelium. Hearing (thirty-six inch watch), right normal; left one and a half inches.

April 3rd, 1900. Left ear remains dry, and hearing continues fair. Conversation is easily heard with right ear closed. Patient has not attended for two months. She described the noises as much diminished in frequency and loudness since operation.

14. P. H. H. P., æt. 24. Seen on November 3rd, 1899. Is deaf in both ears; left worse; history dates to measles at six; with much discharge from left ear until after he left school at eighteen. Tonsils and post-nasal growths were removed in 1888, when the deafness improved, but otorrhœa continued.

Discharge now amounts to moisture in the meatus in the morning, and this troubles patient chiefly, because by passing into the pharynx, it causes his breath to be foul, which is a great annoyance to him in his profession as a dentist. Has not had any pain for some years until this summer, when he has had three severe attacks lasting several days, after each of which "a caseating mass came away" (? cholesteatoma). Patient is now a perfect self-taught lip-reader when addressed on the left side. The deafness in the left ear has not increased for six years; in the right it has been worse during the last year. He has always had tinnitus; much worse on the left side; he is now accustomed to it. Patient has syringed the left ear for varying periods with different solutions, having used carbolic acid and boracic acid, sanitas, and is now using chinosol. Hearing (watch 36 ins.) right, 3; left, 1. Eustachian tubes open to "Valsalva." Right membrane retracted and malleus is immobile to Siegle's speculum. Left meatus full of pus; when cleaned shows discharge from the attic by a perforation in Shrapnell's membrane.

December 27th, 1899.—Otorrhœa has continued in spite of treatment, and tinnitus persists. Left membrane, malleus, and incus removed to-day under A.C.E. The descending process of the incus and the head of the malleus show foci of caries. The packing was removed and changed after the usual intervals, and the ear was quite dry on January 11th (fourteenth day) when the hearing of the left ear had increased to five inches.

On January 18th, when treatment was discontinued, the hearing was ten inches with the thirty-six inch watch, and for conversation and whispering is practically normal; hearing in the right ear has much improved under the use of Politzer's bag. April 19th, 1900, the whole tympanum is quite dry, hearing continues excellent. He is no longer troubled with foul breath.

15. E. H., female, æt. 22. March 21st, 1899. Has had discharge from the left ear for eighteen months. The right ear is normal. On examination of the left meatus there is a large posterior perforation involving both posterior superior and posterior inferior segments. This ear was treated with injections of

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by Removal of the Malleus and Incus.*

boracic acid, carbolic acid, and chinosol lotions, and insufflations of Pulv. Ac. Bor. et Hydrastis. June 12th, no discharge for last six weeks; treatment continued. June 27th, some pain; membrane injected.

October 31st. Discharge, which began again immediately after last note (in June), has again ceased—watch, left ear, 4 ins.

November 21st. Discharge has begun again. January 30th, 1900. Discharge began again on January 9th, after intermission of five weeks, and as patient wished for more active treatment, the membrane, which now only showed a very small posterior superior perforation, was to-day excised with removal of the malleus. Several fragments of the incus were hooked out, and also the head of the stapes and just the outer end of one crus.

February 27th. Tympanum is granulating still, and there is a little discharge from the floor. Patient is to use Lotio Formaldehyd. and Guttæ Sp. Vin. Rect. She had omitted to come for a fortnight, and packing which should have been changed weekly was, therefore, discontinued, owing to the risk of its becoming foul.

A CASE OF ADDISON'S DISEASE, FATAL BY SUPPRESSION OF URINE.

By T. WILSON SMITH, M.D.

THE occurrence of fatal suppression of urine in Addison's Disease is sufficiently rare to merit recording, more especially from the historic interest to Guy's men always attaching to cases of this disease.

Florence M., æt. 15, was admitted to the Royal United Hospital, Bath, on October 13th, 1898. The previous history was that she had been noticed to be getting darker in colour for three months, especially about the face, lips, neck and hands. Till recently her health had been good. The bowels had been confined. Catamenia commenced at fourteen, occurring regularly since. Family history: Her father had died of phthisis.

On admission.—She was a well-nourished girl, with dark, crisp, black hair, suggestive of Creole origin (which, however, was not the case). Her complexion was olive coloured, just as would be usual with such hair, but her mother said that she had been paler and different three months ago. The mucous membrane of the lips was dry, cracked, and pigmented, resembling the effect produced by cauterisation with nitrate of silver. There were also patches or bands of pigment in the mucous membrane lining

the cheeks, but not on the tongue. Opposite to the pigmentation of the lips were two black carious cavities in the upper incisors.

The parts of the arms exposed to the light, and the hands, were darker, as if sunburnt: the usual pressure-marks due to the clothing were no darker than would be expected in a person of the like complexion. Examination of the chest revealed nothing abnormal except that the heart sounds were feeble, nor was anything abnormal felt in the abdomen. The urine was normal until November 7th.

On October 15th and 16th the patient was sick each morning.

October 17th she was better, but had a slight fainting attack whilst coming from the lavatory. She was kept in bed and remained pretty well till November 6th, when her temperature rose suddenly in the evening to 103.4° .

November 7th. Temperature 102.4° . Vomited once in the morning. Tongue furred. There was pain in the abdomen, particularly in the left iliac region; nothing could, however, be felt there.

November 8th. Temperature varied from 100° to 106° . As no urine was voided, a catheter was passed and one ounce of urine drawn off. It was highly albuminous but was accidentally thrown away before it could be further examined. The girl was now vomiting frequently, whenever anything was taken. The pulse and heart's action were very rapid and feeble; no murmur was heard. She became very restless in the afternoon.

On November 9th patient was very ill, pulse imperceptible, hands cold. Vomiting now ceased, but no urine was passed. A vapour-bath was given in which she sweated, but she was hardly conscious and remained very restless, becoming delirious in the evening and dying at 9.40 p.m. She had passed no urine since the morning of November 8th, when one ounce only had been withdrawn by catheter. The bowels had acted on November 8th and several times subsequently.

Post-mortem examination.—Both suprarenal capsules were felt to be hard, the left the more so, containing yellowish nodules in the medulla; the *right suprarenal capsule* was not so hard, and on section was typical in appearance, containing caseous pus

in the medulla. The *kidneys* were swollen, the cortex congested, the pyramids rather pale; there was a small cyst in the cortex of one kidney. The capsule stripped smoothly and readily. The *heart* was small; the other organs quite healthy.

Microscopical examination of the kidneys showed cloudy swelling of the tubular epithelium, and extreme engorgement with blood of the intertubular vessels, with some small extravasations; there was also extravasation of blood cells in the Malpighian capsules.



A CASE OF FATAL HOMICIDAL FRACTURE OF THE LARYNX.

By LOCKHART STEPHENS.

FATAL Homicidal Fracture of the Larynx is far from common in medico-legal experience, and the case here recorded is, as far as I am aware, the only one in which death followed immediately upon the injury being received.

On October 26th, 1895, at 8.30 p.m., I was hastily summoned by a gipsy to his brother who was, he thought, dying on the road-side about a quarter of a mile distant from my house. Upon my arrival, I found the body of a man lying flat upon his back, his head being upon the footpath and his feet toward the middle of the road. The face was livid and cold; the pupils widely dilated; the hands were open and in a natural position with the palms upwards at the sides of the body. Although the night was cold, the chest and abdomen underneath the shirt were quite warm. There was no blood in the nose, ears, or mouth; there were no marks of a struggle in the road. He had no hat, jacket, or waistcoat on. The body was taken by the police, under my direction, to the coach-house of a neighbouring inn, the clothes were removed, and an external examination made. The body was that of a well-nourished and muscular young man. The face was congested and dark. There were no marks of violence upon the body beyond three small abrasions and bruises on the back of

the shoulder as if from scraping against the road. No blood was found upon the clothing beyond a small stain or smear of blood as from an abraded knuckle upon the upper part of the left side of the front of the shirt, and on the right side of the front of the trousers was a dry but recent stain two inches by one and a half inches.

The following account of the way in which the man came by his death is taken from the sworn evidence of witnesses at the inquest, and from the personal statement of the man who caused his death: A party of three gipsies, well known at fairs and race-meetings as rough characters, spent the day together drinking at various inns and finally quarrelled at a public-house in Emsworth. Two wanted to fight each other, and the third endeavoured to act as peacemaker; before they reached the open country they all alighted, left the horse and cart by the roadside, and two began fighting, being stripped to their shirt sleeves. The deceased was a tall (five feet eleven inches) heavy man, his opponent being about five feet six inches and of slight build. Both were well known as fierce fighters, each with a style peculiarly his own, and whilst the former usually attempted to knock his man out by collaring him low beneath the knees, thereby causing him to fall backwards on the ground and strike his head, the latter fought chiefly short-armed blows straight in front of him. The deceased was partially intoxicated, and whilst attempting to carry out his favourite trick was met by his opponent, who well knew his method of attack, with a short, powerful, upper-cut blow. From the description given to me at the time both men must have been in a crouching position, the one attempting to collar the other, when the gradually stooping weight of his body was met by the strong upward blow of the other. Immediately on receiving this the man threw up his arms and toppled over backwards, probably dead, as he was not seen to move afterwards.

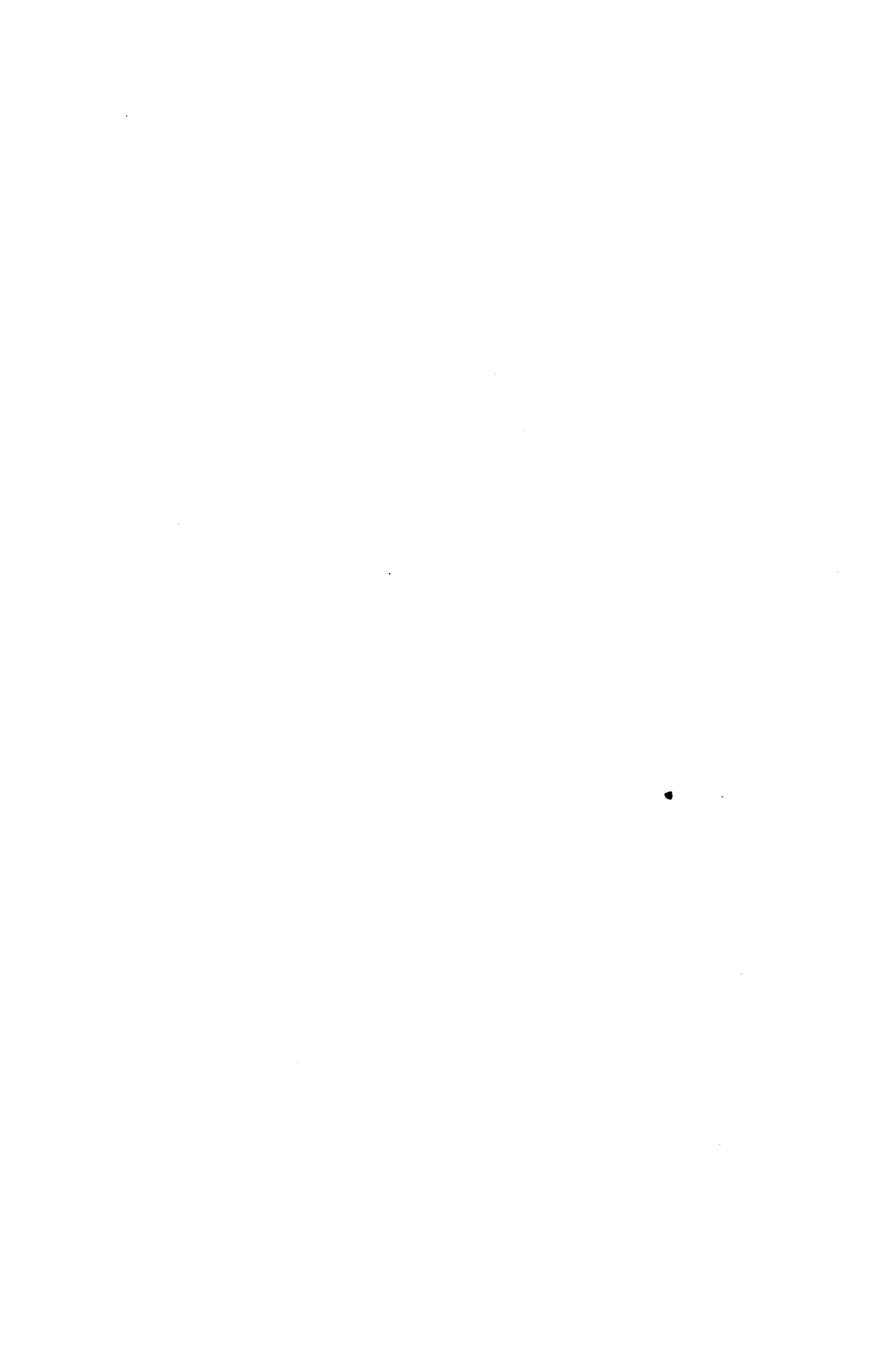
A *post-mortem* examination was made forty-two hours after death. Rigor mortis well marked, no subconjunctival ecchymoses. The hyoid bone could be felt to be broken, but there was no effusion of blood into the tissues or muscles of the neck. The tongue, larynx, and trachea were removed *en masse* for further

examination. Nothing abnormal was found in the chest, abdomen or cranium, the contents of which were most carefully examined. There was no fracture of the skull. At this stage of the post-mortem I unfortunately punctured my finger, and so packed up the specimen (tongue, larynx, etc.) and together with the cervical spine and heart sent them to Dr. J. H. Targett, the then curator of the Pathological Museum, R. C. S., London, who reported to this effect :—

After maceration of the spine no trace of fracture was present, the transverse ligament was not ruptured, and the cord showed no trace of laceration or hæmorrhage. As regards the fracture of the larynx, the right great cornu of the hyoid bone was broken close to the body and there was a little hæmorrhage around it. The superior cornu (left) of the thyroid cartilage was also snapped off, and there was a little blood round it which showed through on the mucous surface of the pharynx.

The great point of interest in the case is, Why did the man die? There was absolutely nothing abnormal found post-mortem with the exception of the fractured larynx. The question therefore resolves itself into—

Can sudden death arise from a fractured larynx?



SOME REMARKS ON SURGICAL INTERFERENCE IN GASTRIC ULCER BEFORE AND AFTER PERFORATION INTO THE PERITONEAL SAC.

BY A. R. THOMPSON.

At the beginning of this paper it is necessary to discuss briefly the pathology of gastric ulcer, the exact causation of which is not known, but a local necrosis owing to the presence of thrombi in blood-vessels may be the means whereby an ulcerative process is started. Gastric ulceration occurs more frequently in women than in men, and more often attacks women between the ages of twenty and thirty, men between the ages of thirty and forty, than at other periods. The ulcer in the majority of instances occurs on the anterior surface, and at the pyloric end of the stomach.

Professor Osler, in his text-book of Medicine (Third Edition, 1898, p. 478) says that ulceration of the stomach is usually associated with hyperacidity of the gastric secretion.

The ulcer starting in the mucous membrane of the stomach tends to perforate the various coats of that viscus, becoming smaller in diameter as it approaches the serous coat; it is thus funnel-shaped, the apex looking outwards. The edges of the ulcer are at first regular, rounded, and smooth; but if the ulcer has existed for some time, the edges become thickened, indurated, and irregular, and peritoneal adhesions to structures adjacent to the ulceration may be formed.

Gastric ulcer may heal of its own accord, or may be cured by medical means, but its tendency to perforate, if not cured, either into a large blood-vessel or into the peritoneal cavity, marks the condition of ulceration as a dangerous disease.

As the most important result of progressive ulceration is perforation into the peritoneal sac, this aspect of the question may be considered first.

Actual perforation of the stomach-wall will give rise to acute or chronic symptoms: to acute symptoms when the general peritoneal sac is placed in communication with the cavity of the stomach; to chronic symptoms when only a small portion of the peritoneal sac is open to the escape of gastric contents.

PERFORATION INTO THE GENERAL PERITONEUM, OR ACUTE PERFORATION.

The symptoms of acute perforation are: a history of something having given way inside, usually after some exertion, accompanied by a sudden sharp pain which doubles up the patient, being so severe sometimes as to cause fainting. The pain is usually described as situated in the region of the stomach, *i.e.*, in the epigastrium and left hypochondrium, but it may be referred to the umbilicus, and, later, even to the iliac fossæ. The pain may also be general. The pulse becomes rapid and feeble, the breathing quick and thoracic. The temperature varies, it may be normal, subnormal, or above normal. The knees are drawn up, and the abdominal muscles are rigid. Vomiting may occur.

As these symptoms pass off the patient begins to improve, this being, perhaps, due to the fact that sedative drugs have been administered, but also, perhaps, to the reaction of the tissues.

This reactionary period is quickly followed by symptoms of peritonitis and collapse, when the abdomen becomes distended and hard, and the liver dulness as a rule disappears, a fact due to the presence of gas from the stomach itself, or to gas produced, as Mr. Eve thinks, by decomposition (*vide Lancet*, vol. i., 1900, January 20th). Perforation having been diagnosed, the question of operation arises.

IS OPERATION INDICATED IN ACUTE PERFORATION?

Dr. Habershon, in his book on "Diseases of the Stomach," p. 248, says the chances of recovery are slight. This remark was made before surgical interference was suggested by Mr. Dobson, of Bristol.

Greig Smith, in his work on Abdominal Surgery, says of cases unoperated upon, that 90 per cent. are fatal. The writer has collected one hundred and three cases recorded in the journals, and finds that after operation 60 per cent. recovered, but recorded cases are very fallacious, and it became necessary to look up hospital records. For this purpose the records at Guy's Hospital and the London Hospital, as well as the official catalogues of the Leeds and Bradford Infirmaries were consulted. In addition to these important sources of informatjon, there are included in the table given below, seven cases recorded by Mr. Barker in a clinical lecture (*Lancet*, vol. ii., 1896, p. 1583), and seven cases recorded by Mr. Maurice, of Reading, as occurring to his knowledge (*Lancet*, vol. ii., 1895, p. 981). The following table has been compiled from these sources.

Guy's, 1885 to 1900	21 cases	16 deaths	5 recoveries.
London, 1893 to 1899	10 cases	9 deaths	1 recovery.
Leeds, 1880 to 1898	4 cases	4 deaths	0 recoveries.
Bradford, 1893 to 1898	3 cases	3 deaths	0 recoveries.
Mr. Barker's cases	7 cases	4 deaths	3 recoveries.
Mr. Maurice's cases	7 cases	4 deaths	3 recoveries.
Total	52 cases	40 deaths	12 recoveries.

Judging from these statistics a trifle over 20 per cent. of cases recovered after operation.

It will be observed that the statistics differ widely from those which were taken from cases recorded in the journals, but it may be noted that, whilst recorded cases err on the side of recovery, hospital cases err on the side of fatal results. This remark is made because the patients' friends have preferred to wait until a condition that seems to them serious is reached, namely, the condition of collapse, before the patient is sent up to hospital. Again, the mere fact of being brought up to hospital necessarily entails considerable movement on the part of the patient.

In addition to this, the patient may be considerably shaken whilst being carried to the operating table.

It may, therefore, be fairly concluded that hospital statistics place the chance of recovery too low. But even if the recoveries are only 20 per cent. of all cases operated upon, this is an appreciable improvement on medical treatment, and it may be therefore urged that surgical interference is indicated when perforation has taken place into the general peritoneal sac, provided always that the collapse is not so great that operation will only hasten the end.

TIME OF OPERATION AFTER PERFORATION.

The operation must not be done immediately after the perforation, because the patient is in a considerable state of shock (Mr. Pearce Gould, *B. M. J.*, vol. ii., 1894, p. 859). The following figures, compiled from the writer's statistics, indicate that operation should be performed as soon as possible after that shock has passed off, as they show that patients have five times more chance of recovering if operated upon within twelve hours after the perforation than they have if operated upon when twelve hours or more have elapsed.

In eleven recorded cases in which the operation was performed under four hours after the perforation, there were eight recoveries and three deaths.

In seventeen cases in which operation was performed between four and eight hours after the perforation, there were thirteen recoveries and four deaths. In ten cases in which operation was performed between eight and twelve hours after the perforation, there were eight recoveries and two deaths. In eleven cases operated upon between twelve and eighteen hours after the perforation, there were six recoveries and five deaths. In twenty-seven cases where the operation was performed, an interval of more than eighteen hours having elapsed, there were seven recoveries and twenty deaths.

As has been pointed out, a reactionary period occurs after the shock, and the patient shows a marked improvement. This is the period when operation should be undertaken, but unfortunately it has proved, in some cases, that the friends of the

patient, and, it is to be regretted, the medical man as well, have been so impressed that operation is delayed ; with the result that collapse and purulent peritonitis almost invariably set in, leading to a rapidly fatal termination.

The conclusions, therefore, that may be drawn from the foregoing statements are :—

1. Shock should be allowed to pass off ;
2. Operation should be performed immediately shock has passed off.
3. A reactionary period occurs and should be carefully noted.
4. Operation should be performed during the reactionary period.

Special attention should be paid to the condition of the pulse, which, as a rule, increases in rapidity as collapse sets in, and any increase in rapidity must at once be considered as indicating an operation. Mr. Symonds, however, told the writer that he had seen a case where the pulse did not become more rapid than sixty-eight per minute.

If the operation is going to be performed in private practice, the patient must not be removed from the room in which the perforation occurred, and if in a hospital, as little movement as possible of the patient should be allowed, both before and after the operation.

The patient should be laid on a warm table and covered sufficiently with flannels. The limbs should be swathed in cotton-wool and bandaged, and every detail should be carried out as aseptically as possible. The patient having been placed under the influence of an anæsthetic, an incision is made in the middle line between the ensiform cartilage and the umbilicus for a length of four or five inches, as by this median incision all parts of the stomach may be more easily explored than by other incisions. If the operator, however, finds that he has not sufficient room to view the stomach in all parts, even after retracting the sides of the wound as far as possible, he may make a transverse incision across the rectus muscle, usually the left ; but there are some objections to this step, namely, that by cutting across the arteries and veins much bleeding is caused, a most

undesirable occurrence considering the critical state of the patient, and also that by transverse division of the arteries and nerves a weak scar results after the wound has closed up.

When the peritoneum is reached it, in some cases, bulges owing to the presence of gas, but in two cases operated upon by Mr. Symonds in Clinical ward, which the writer was fortunate in seeing, there was no bulging at all. When the peritoneum is cut through, gas and gastric contents escape. The gastric contents should be carefully sponged away in order that the orifice through which they escaped may be discovered. If this orifice be on the posterior surface of the stomach it will not be seen, but if on the anterior surface, gastric contents will usually be observed welling through the perforation. If the orifice be discovered, further escape of the contents of the stomach must be prevented, either by washing out the stomach, or by closing the perforation temporarily either with the fingers, or by plugging, or by temporary sutures passed through the stomach wall.

If, on the other hand, the orifice is not at once seen, much difficulty may be experienced in finding it. It may be altogether out of sight, or it may be so minute as to escape notice; in the latter case, as suggested by Dr. Hale White (*B. M. J.*, May 18th, 1895, p. 1094), the stomach might be blown up with gas or fluid through the mouth. For the purpose of methodically exploring the stomach, the following table compiled by the writer may be found useful by operators.

Out of ninety cases of perforation into the peritoneal sac, collected from the journals and the records at Guy's—

86	occurred on the anterior surface,
11	" on the posterior surface,
32	" at the cardiac end,
19	" at the pyloric end,
8	" on the lower curvature,
2	" on the greater curvature,

and in three cases perforation occurred on both anterior and posterior surfaces.

[NOTE.—The writer must point out here that if perforation occurred, *e.g.*, on the anterior surface and also at the cardiac end, it is included twice in the table.]

If, therefore, no orifice is discovered at once, it is clear that the anterior surface should first be carefully examined and at the cardiac before the pyloric end. The lesser or greater curvature should then be examined according as to whether the posterior surface is to be approached from above or below; if from above the lesser curvature should be examined first, and then the gastro-hepatic omentum torn through, and the stomach turned down, a proceeding which is attended with much danger to important vessels. If, on the other hand, the posterior surface be exposed from below, the greater curvature should first be examined and the anterior layers of the great omentum torn through and the stomach turned up. Another method of exploration is recommended by Mr. Wallis (*B. M. J.*, vol. ii., 1898, p. 1686), namely, that a gastrotomy should be performed and the stomach explored from within.

The presence of adhesions may cause some delay in finding the perforation, though they may indicate its position roughly. So far as the writer's enquiries go he is convinced that adhesions should be broken down in order to expose the perforation. In all the cases at Guy's Hospital where the perforation was not found, owing to the presence of adhesions, death occurred.

If the operator cannot see the perforation at all, Mr. Makins (*B. M. J.*, vol. i., 1897, p. 914) recommends judicious palpation, as it may happen that the ulcer is indurated, and thus possible to feel. If necessary, the stomach may be drawn into the wound in order to discover the perforation.

The ulcer having been discovered and secured temporarily, the next step is to secure it permanently. After separating the ulcerated area from neighbouring structures by gauze tampons wrung out in sterile water, the following plans are open to the surgeon, according to the state of the patient and the condition of the ulcer.

1. Excision of the ulcer.
2. Suture with inversion of the ulcer.
3. Production of a gastric fistula.
4. Introduction of a Paul's tube.
5. Use of an omental plug.

1. Excision of the ulcer.—This plan is adopted by some surgeons, and doubtless is of use when the ulcer is not one which has lasted for some length of time, in which case the tissues adjacent to the perforation do not lend themselves readily to suture, especially when the tissue is soft and stitches are apt to break through. But valuable time may be thus spent which might be devoted to more important details, and excision may convert the orifice of the perforation into a larger gap, the edges of which the surgeon may find some difficulty in bringing together. An interesting case is recorded by Mr. Paul Swain (*Lancet*, vol. ii., 1894), where, owing to the excision of the indurated area round the perforation, the total ulcerated area being two inches in diameter, a large gap six inches in diameter resulted owing to the relief of tension and the normal elasticity of the gastric muscle; thirty-five sutures were used for bringing the edges together.

2. Suture and inversion of the ulcerated area.—This step is performed by passing sutures through the whole thickness of the stomach wall close to the perforation, and then by passing Lembert's sutures outside the ulcerated area in the healthy tissue of the stomach, to turn the ulcerated area into the cavity of the stomach. There is a most interesting case recorded by Mr. Alexis Thomson where this was done, and at the necropsy (death taking place from pneumonia ten days after the operation) no signs of ulceration could be seen, the whole of the inverted area having been devoured by the action of the gastric juice (*Lancet*, vol. ii., 1896, p. 11).

3. Gastric Fistula.—This step is undertaken when the edges of the orifice are too far apart to be conveniently brought together.

The edges of the orifice, after being freshened, are brought up to the wound in the abdominal wall and stitched there. The objections are—

1. That by this means free cleansing and drainage of the peritoneal sac is rendered difficult.

2. That extreme difficulty may be experienced in closing the fistulous opening.

But, on the other hand, nourishment may be introduced into the stomach through the opening—a practice which cannot be adopted when the perforation is closed up immediately.

4. Greig Smith reports a case of Mr. Paul's, where the latter surgeon introduced his tube into the opening in the stomach, and had no difficulty in making the orifice close.

5. Another method first suggested by Mr. Bennett (*Lancet*, vol. ii., 1896, p. 310) may be adopted when the orifice is too large to be closed by bringing the edges together.

This consists in bringing up a piece of omentum and plugging the orifice with it, and then, after transfixing the plug with one stitch, attaching it to the edges of the orifice by Lembert's sutures, thus bringing the surfaces of peritoneum together.

The late Mr. T. Jones, of Manchester, has suggested a method of bringing up a piece of omentum and stitching it over a perforation which has been previously closed up in either of the first two methods (*Lancet*, vol. i., 1899, p. 95). This plan has been carried out with considerable success.

CLEANSING OF THE PERITONEUM.

This question is at present the subject of much discussion, as some surgeons advocate irrigation and some simple sponging. At a meeting of the Clinical Society, held on December 9th, 1899, which the writer was enabled to attend through the kindness of Mr. Symonds, most of the speakers who discussed the detail of cleansing the peritoneum were in favour of irrigation, but some advocated carefully sponging the peritoneum. It will be, therefore, well if this point is discussed here.

A. Irrigation.—The fluid used for irrigation should be sterile water, normal saline solution, or some weak antiseptic. Whatever fluid is used should be heated to 105° F. at least; but it may be here observed that irrigation is not used by surgeons for the purposes of antiseptics, but as a mechanical means of aiding the peritoneum to dispose of the gastric contents, this serous membrane having very considerable power to dispose of foreign material satisfactorily, a statement proved by the fact that perforation having undoubtedly occurred, recovery has followed

without surgical interference, and also by a case reported by Mr. Littlewood (*B. M. J.*, vol. ii., 1896, p. 1324), in which that surgeon is certain he left some foreign material in the peritoneal sac, the patient making a good recovery.

It may be that by irrigation particles of food will be driven into the fossæ and pouches of the peritoneum, in such a manner that no amount of irrigation will dislodge them, and imperfect cleansing or even additional failing may thus be a result.

But in the two successful cases mentioned above and operated upon by Mr. Symonds, irrigation was practised, and if irrigation be methodically performed, as it was in these two cases, there is no more danger of inadequately cleansing the peritoneum than by other means adopted for clearing out foreign material.

In the majority of the cases which the writer collected when observations were made, much food was found in the pelvis, and in order that this part of the abdominal cavity may be thoroughly cleansed, a suprapubic incision is frequently made and the pelvis cleaned through this aperture in the abdominal wall. But whether the plan of making the suprapubic incision be adopted or not, too much stress cannot be laid on the fact that the irrigation, if it is to be used at all, must be used methodically.

The nozzle of the irrigator should be directed all over the peritoneum, beginning at the lowest part of the ascending colon and passing up to the liver, paying especial attention to the upper surface of that organ, and then across the abdomen, passing the liver, stomach, and small omentum, to the left side, where again attention is paid to the upper surface of the liver. From thence the irrigating process should be pursued down the left side of the abdomen, along the descending colon, special precautions being taken to thoroughly cleanse the pouches above the gastrophrenic ligament and the phrenocolic ligament, and from thence down to the pelvis.

Irrigation should be continued until the fluid leaving the cavity is perfectly clear. The abdomen may then be mopped out with dry sterile sponges.

B. Sponging the peritoneum.—This method is adopted by some surgeons. The peritoneum is carefully sponged, the bowels

being brought out through the wound, if necessary, and the finger-nail cautiously used for removing lymph and foreign material. Though this method may be less liable to leave behind collections of food, yet it takes a longer time than irrigation; and it is desirable that the operation should be as short as is compatible with performing thoroughly the cleansing of the peritoneum.

From the cases the writer collected when a definite history was given of the means adopted for cleaning the peritoneum, the following figures are noteworthy:—

In eleven cases where irrigation and sponging were adopted, there were six recoveries and five deaths;

In nineteen cases where irrigation was practised alone, there were eleven recoveries and eight deaths;

In sixteen cases where sponging alone was adopted, there were fourteen recoveries and two deaths.

It is thus shown that, as far as these figures can be relied on, the last method is better than the others, and that efficient cleansing depends on the method adopted as well as on the care displayed by the surgeon in the details of the method.

It cannot be too strongly urged here that the whole operation is rendered useless unless most thorough and patient steps are taken to clean the peritoneum and its pouches, especially above and behind the liver.

Irrigation of the lesser sac.—In two cases subphrenic abscess occurred in connection with the lesser sac when the perforation was in the anterior surface, one reported in the *Lancet*, vol. i., 1899, p. 95, the other reported in the *Lancet*, vol. ii., 1898, p. 1761; both the late Mr. T. Jones, of Manchester, who operated in the former case, and Mr. Willoughby Furner, who operated in the latter case, advocated opening up the great omentum and cleansing the lesser sac, but, on the other hand, it may be stated that the foramen of Winslow may be closed very easily by adhesions, and certainly in one case recorded where it is definitely stated that the lesser sac was not cleansed the patient made a good recovery.

It may be said, therefore, that the surgeon must use his own judgment in deciding whether the lesser sac should be irrigated

or not, bearing these two facts in view: first, that he may infect a clean sac, and secondly, that adhesions may have formed at the foramen of Winslow. If, on the other hand, foreign material has obviously entered the lesser sac, especially when the perforation is on the posterior surface of the stomach, it is absolutely necessary to thoroughly cleanse the lesser sac.

The wound may be closed altogether or only partially sewn up and a drain inserted. The drains should be of gauze, indiarubber tubing, or glass, the last being the most satisfactory, as suction action may be more conveniently performed if this material is used for drainage.

After the operation the patient should be removed carefully back to the bed, which must be warm.

No food should be given by the mouth for three or four days, and, if possible, no water should be administered, the excessive thirst caused by depriving the patient of water being relieved by axillary injections; but if these do not avail, then water must be administered to the patient, a teaspoonful at a time being allowed.

At the end of three or four days, water may be allowed in larger quantities, and then milk, and, by increasing the diet gradually, ordinary food may be eventually taken.

It will be useful here to point out the causes of death after perforation where an operation was performed, but failed to avert death.

Death due to general peritonitis, either as a result of the perforation, or insufficient cleanliness on the part of the operator, is not included, as the writer felt that in some cases it is difficult to discriminate between contamination before or after the operation.

The perforation, though present, was not found in thirteen cases of the total number the writer has notes upon, viz., one hundred and twenty-two; of these thirteen cases, eleven died, shewing that the perforation must be found at all costs, even to breaking through adhesions and healthy peritoneum. Localised abscesses occurred in twelve cases out of one hundred and twenty-two. In ten of these cases the abscess was subphrenic, and situated as

a rule on the left side of the falciform ligament. In two of these cases the abscess was perisplenic.

Death took place, on an average, in these cases in about a fortnight after the operation, the longest interval between operation and death being six weeks. In only one case did death take place in so short a time as one day after the operation, and this was due to an abscess bursting and discharging into the general peritoneal sac, giving rise to symptoms of perforation of the stomach.

It will thus be seen that as a rule a localised abscess does not cause rapid death. Its causation is due to a localised inflammation, adhesions being formed before actual perforation takes place, or it is due to stagnation of foreign material setting up a localised peritonitis with a formation of adhesions, and so the foreign material is enclosed, giving rise to a local suppuration.

Abscesses occurring anywhere in the peritoneum must, of course, be treated at the discretion of the physician who diagnoses and of the surgeon who operates, but in the case of a subphrenic abscess resection of the 8th rib, usually on the left side, may be adopted, and the abscess drained by that means, as in a case of Mr. Dean's at the London Hospital.

Localised pelvic peritonitis does not often occur, but this is due to the fact that operators realised early that the pelvis was a most important position to attend to in cleansing the peritoneum, and took care that imperfect cleansing of that portion of the abdominal cavity should not be responsible for the patient's death.

In three cases perforation into the peritoneal sac occurred in connection with an ulcer on the posterior surface after perforation of an ulcer on the anterior surface had been sewn up.

This fact may be explained by the breaking down of protective adhesions, binding the posterior surface to neighbouring viscera owing to the handling of the stomach, and allowing communication between the gastric cavity and the peritoneal sac; and the same fact may be pleaded as a reason for excision of the edges of a perforation in all cases, whether indurated or not, in order

to examine the interior of the stomach with a view to discovering a second ulceration.

THE QUESTION OF OPERATIVE INTERFERENCE IN HÆMATEMESIS.

Hæmatemesis is a very frequent symptom in gastric ulcer; the bleeding may arise from a very small ulceration only involving the mucous membrane, such an ulceration sometimes occurring in connection with backward pressure upon the veins of the stomach; or it may take place from an ulcer involving other coats of the stomach, in which case the bleeding may take place from a large vessel which has become perforated by the ulcerative process. It is obvious that if there is some constitutional disease leading to backward pressure upon the veins of the stomach over which ulceration has taken place, surgical interference will be of little use; but if the lesion be not due to such back-pressure, surgical interference has been undoubtedly beneficial in certain cases.

Hæmorrhage from the stomach is of three kinds—acute, sub-acute and chronic.

Acute hæmorrhage occurs when some large vessel is opened up by the ulcerative process, leading to immediate death. Subacute hæmorrhage occurs when a smaller vessel is opened, as in a case under the care of Mr. Symonds, in October, 1899, when the bleeding took place from the pancreatico-duodenal artery at the pylorus, and death occurred in not less than five hours from the onset of symptoms of hæmorrhage.

Chronic hæmorrhage is the most frequent of the three varieties, and arises from the raw surface of an ulcer irritated by the passage of food. In the acute cases of hæmorrhage, when some large vessel is opened, death occurs so soon that there is no time for surgical interference; but in the subacute and chronic types of hæmorrhage this statement cannot be pleaded as an excuse for withholding interference, provided any of the following indications are present, viz., when the patient is showing grave symptoms of collapse from hæmorrhage, there being a previous history of ulcer of the stomach; or when more than one litre of blood has been brought up at one time (Von Leube); or when hæmatemesis so frequently recurs that the patient becomes anæmic and wasted.

Surgical interference having been decided upon, it remains for the writer to try and indicate the origin of bleeding, as the operator may find some difficulty in locating the bleeding point, owing perhaps to the small size of the ulcer, or to the fact that the stomach may be filled with blood.

The following figures were tabulated by Savariaud :—

Hæmorrhage of Venous origin	4
" Arterial origin	47
" from Splenic artery	17
" " Coronary artery	6
" " Arterioles	10

It will be seen that in seventeen cases bleeding took place from the splenic artery; but this artery is a vessel of considerable length, and a more exact indication of position is necessary than that afforded by a mere statement of the source of bleeding. The writer therefore collected certain cases (*vide* appendix) from the journals and post-mortem records at Guy's (fourteen in number), out of which nine cases of hæmatemesis were from an ulcer in the region of the pylorus, in three of which an arterial source was obvious; in only one case was there bleeding from an ulcer situated on the anterior surface. Andrew's and Eisendrath's statistics confirm the writer's observations. Out of nine cases collected by these authors, seven cases of hæmatemesis occurred in connection with a pyloric ulcer, and not one is recorded by them in connection with an ulcer on the anterior surface. The pyloric region should, therefore, be first thoroughly explored by the surgeon with a view to locating and arresting the hæmorrhage, for the accomplishment of which object several methods are open.

If the hæmorrhage occurs in association with ulceration of the mucous membrane, a gastrotomy may be performed, and the ulceration under-run with silk and ligatured, as in the following case which Mr. Mansell Moullin has kindly allowed the writer to report. A patient was admitted into the London Hospital under the care of Mr. Mansell Moullin, for weakness, emaciation, dyspepsia and recurrent hæmatemesis of some years' duration. On performing a gastrotomy, small superficial mucous ulcerations were seen at the

cardiac end and under-run with silk and tied. Complete recovery with cessation of symptoms followed. In other cases, a gastrotomy having been performed, the ulcerated area may be gently pulled or pushed into the cavity of the stomach, and the base of the cone thus formed securely ligatured (Andrews and Eisendrath).

If the bleeding be due to an ulcer involving more than the mucous membrane of the stomach, any of the following plans may be adopted :—The arteries supplying the area of ulceration may be ligatured—Mr. Openshaw of the London Hospital was the first surgeon to adopt this method in a case where the gastro-duodenal and coronary arteries were ligatured ; the main objection to this method of operation being that as the anastomosis of vessels supplying the stomach is very free, arrest of hæmorrhage may not be accomplished unless all the arteries of the stomach are ligatured, a proceeding which is quite impermissible.

Andrews and Eisendrath have performed, with success, a gastrotomy, and then inverted the ulcer, ligaturing it tightly at the base. Mr. Tubby also records a case where the ulcerated area was pulled out and ligatured tightly at the base, but the piece of tissue thus treated is liable to set up a peritonitis if it becomes necrosed, an occurrence which, in this case, had not taken place after a lapse of ten days, when the post-mortem examination was made, the patient having died from collapse (*Lancet*, vol. i., 1899, p. 1267).

A modification of the previous methods has been adopted, in which the arteries chiefly supplying the area of ulceration are ligatured ; and the ulcer then excised or inverted.

Küster has, in addition, adopted another method of arresting hæmorrhage :—

A gastrotomy having been performed, the actual cautery is applied to the bleeding area, and a gastro-enterostomy then performed, thus placing the ulcer at rest, and facilitating healing ; further, this step disposes of any increase of the trouble which may be caused by cicatrisation of the ulcer, as owing to the gastro-enterostomy the continuity of the alimentary tract is not affected by the stenosis which follows cicatrisation.

Mr. Mayo Robson has performed (New Year, 1900) at least two cases of gastro-enterostomy with complete cessation of hæmorrhage.

OTHER CONDITIONS CALLING FOR SURGICAL INTERFERENCE.

Although perforation and hæmorrhage are serious complications of gastric ulcer, it may be doubted whether, as far as the patient is concerned, they are of more importance than the complication which is immediately to be considered, namely, progression and cicatrisation of the ulcer leading to obstruction in the alimentary tract, either by actual narrowing of the lumen of the bowel, or by the formation of peritoneal adhesions leading to constriction.

The position of an ulcer most often giving rise to obstruction is at the pylorus, and this may be expected, as the ulcerative process is occurring at a narrow portion of the gut. Another position where a healed or progressive ulcer has caused obstruction is on the lesser curvature leading to the formation of an hour-glass stomach, but even if such a condition gives rise to symptoms calling for surgical interference it may be doubted, on anatomical grounds, whether operation could be performed likely to relieve the patient, unless heroic measures were undertaken. Gastro-enterostomy might be performed on the œsophageal side of the constriction, but in some cases this would hardly be possible, as the constriction may not be far away from the cardia.

As regards the pylorus the condition is quite different, as the bowel is here narrow and easily accessible; and in order to get rid of the evil consequences following upon pyloric ulceration and stenosis, any of the following operations may be performed.

- A. Gastro-enterostomy may be adopted. The advantages of this method are :—
1. Shortness of time consumed in operation, a desideratum always, but especially so when the patient is in a weak condition. Gastro-enterostomy may be performed in twenty-five minutes.
 2. It places the ulcer at rest surgically—a condition aimed at by every physician—and allows it to heal, and even if cicatrisation and stenosis at the site of the ulcer takes place, no bad effect can follow upon the passage of food from the stomach to the intestines.

3. It may be carried out where there is cartilaginous induration about the pylorus not allowing of pyloroplasty or Loreta's operation.

Mr. Symonds, at a meeting of the Hunterian Society, said that in some cases a gastro-intestinal opening became contracted. This being so, it is a serious objection, but may be overcome by making the opening in the stomach oval, a proceeding advocated by Mr. Symonds.

B. Excision of the ulcer. This has been adopted, but the objections to it are :—

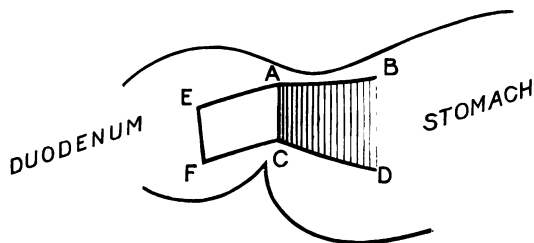
1. The large gap left after excision.
2. The necessary narrowing of the lumen of the bowel.
3. The length of time of operation if the ulcer is large, and it must be remembered that it is a large rather than a small ulcer that gives rise to obstruction.

One advantage is that excision gets rid of the cause of stenosis, but it is very improbable that this counterbalances the objections.

We report one case by permission of Mr. Littlewood, of Leeds, where the large gap left was filled up by stomach wall.

A woman, aged 35, was admitted into the Leeds Infirmary at the beginning of the present year (1900), with a history of vomiting a large quantity of gastric contents at intervals of twenty-four hours or more, and progressive weakness and emaciation. On examination, the stomach was found to be dilated to such an extent that its lower edge was situated two inches below the umbilicus. A median laparotomy was performed, when a thick fibrous adhesion was found attaching the pylorus to the liver. This adhesion was ligatured and cut through. A large indurated ulcer, two inches long and one inch broad, was found on the anterior surface of the pylorus. The ulcer was excised, and a rhomboidal gap left, the edges of which could not be brought into apposition without causing serious stenosis at the pylorus. From each of the angles at the gastric extremity of the rhomboidal gap, an incision was made for a length of two inches into the stomach, the incisions slightly diverging from each other ; a flap of stomach-wall was thus formed and brought down so as to occupy the gap

left by excision of the ulcer, the edges of which were stitched to the edges of the flap taken from the stomach wall, thus—



The shaded area ABCD is the gastric flap; the points A and C were brought down and stitched to E and F.

The patient made a good recovery, and within two months had gained weight, and the stomach had regained nearly its normal size. There was complete cessation of vomiting.

C. Pyloroplasty. This operation is performed by making an incision longitudinal to the pylorus, through the cicatricial area, and extending the incision on each side of the stenosis. This longitudinal cut is converted by traction into a transverse incision and sewn up as such.

If the ulcerated area be small, a longitudinal cut may be made over the ulcer, and the edges of the incision pared away, so as to form a wedge-shaped gap, the edges of which are brought together in such a manner that a transverse slit is formed, the sides of which are sewn up.

It may be stated that the operation is only indicated in cicatricial stenosis, since a progressing ulcer, unless small, is not removed by it, nor does it in any way put the ulcerated area at rest.

D. Loreta's operation for cicatricial stenosis.

After reading of this operation, and bearing in mind the properties of fibrous tissue, the writer felt that probably all recoveries which occurred, took place in spite of the surgeon's interference, and he sees no reason to go back from that opinion. The operation

consists in performing a gastrotomy, and dilating the pylorus by means of graduated dilators, beginning with a pair of dressing forceps or a catheter, and going on with the fingers. Loreta himself used to dilate up to three fingers.

Undoubtedly cases have occurred where complete relief has been obtained, but the following objections on its pathological and surgical side may be considered :—

1. Cicatricial tissue consists of fibrous tissue. Fibrous tissue is not extensible to anything, save to a slight degree. In attempting, therefore, to dilate a cicatricial stenosed opening, either rupture must occur, or stenosis will return owing to the nature of the tissue.

It was argued that as dilatation of a strictured rectum was followed by good results, that dilation of a pyloric stricture would be followed by equally good results, but the conditions are different.

The rectum may be kept dilated, the pylorus cannot.

The rectum is surrounded, at any rate where stricture usually occurs, namely, in the anal region, by tissue composed of fat and muscles. Even if rupture does take place, no very serious result can occur, but if rupture takes place in the duodenum, rapid death is almost certain to occur.

2. The operation is not permanent.

3. It gives rise to rapid death occasionally.

4. It is of no use for a progressive ulcer, as during dilating the opening, rupture may occur, or such disturbance of the ulcer takes place, that fatal hæmatemesis results.

5. It has all the dangers attendant upon abdominal section and gastrotomy, with none of their good results or permanent benefit.

If adhesions have formed due to old ulceration, and they are giving rise to symptoms of obstruction, they should be cautiously divided. Mr. Mayo Robson has recorded two cases where adhesions had formed at the pylorus, leading to dilated stomach, a condition which was cured by division of the adhesions.

Greig Smith, in his book on Abdominal Surgery (q. v.), has pointed out that ulceration on the posterior wall leading to adhesion of the stomach to neighbouring organs, has caused

dilation of the stomach, and such troublesome dyspepsia, that surgical interference is warranted, in which case the ulcerated area should be removed by excision and the adhesions divided.

SURGICAL TREATMENT AS A PROPHYLACTIC MEASURE IN GASTRIC ULCERATION.

It has been shown previously that the great majority of perforating gastric ulcers occur on the anterior surface of the stomach. In addition to this statement, the following figures were collected by the writer from the post-mortem records of Guy's Hospital. Between the years 1888 and 1898 inclusive, there were twenty-five cases of perforation, the ulcer in nineteen cases being situated on the anterior surface. Between the years 1888 and 1898, there were twenty-nine cases of ulcer on the anterior surface, twenty of which had terminated in perforation; that is to say, that more than two-thirds of all ulcers occurring on the anterior surface had terminated fatally. These figures, in spite of the fact that they are taken from post-mortem records, afford good evidence that the position of an ulcer in the stomach alters the prognosis; that, in fact, the prognosis instead of being favourable as in most cases of gastric ulcer, becomes distinctly unfavourable if the ulcer happens to be on the anterior surface of the stomach, and it may indeed be stated that if an ulcer occurs on the anterior surface, there is more likelihood of it leading to a disastrous termination by perforating, than a safe termination by healing. The diagnosis, therefore, of gastric ulcer is not sufficient, but must be further amplified by a statement as to the position of the ulcer, which may to some extent be diagnosed by attention to the following points.

Abdominal Pain.—The pain of pyloric ulcer and duodenal ulcer is situated to the right of the middle line of the abdomen, that of ulcer in the main part of the stomach is situated on the left of the middle line.

The pain of ulcer in the pyloric region occurs later after taking food than the pain of ulcer in the cardiac region. The cutaneous tenderness due to ulceration is on a different plane, according as the ulcer is situated at the pyloric or cardiac end, for the cutaneous

area corresponding to the cardiac region is that supplied by the sixth and seventh dorsal nerves, whilst the area corresponding to the pyloric region is supplied by the ninth dorsal nerve, and the area corresponding to the part between these regions is supplied by the eighth nerve.

Shoulder Pain.—Ulceration on the anterior surface of the stomach may cause pain in the left shoulder, ulceration on the posterior surface may cause pain in the right shoulder. This may be explained as follows: the vagi nerves communicate with the spinal accessory nerves which communicate by means of the subtrapezial plexus with the cutaneous nerves of the acromial region, namely, the third and fourth cervical. As the left vagus supplies the anterior surface of the stomach, disturbance of this nerve by ulceration may cause left shoulder pain, ulceration on the posterior surface causing right shoulder pain.

Vomiting occurs later after taking food when the ulcer is situated at the pyloric end than when the ulcer is situated at the cardiac end.

These statements are founded on anatomical and clinical facts, and have been made as the writer considered that the diagnosis merely of the disease was insufficient unless the position also was localised. Localisation, it may be repeated, is important, as it has been shown that perforation is frequently associated with ulcers occurring on the anterior surface; and if perforation occurs, the patient has a not very hopeful chance of recovery. It may, therefore, be fairly argued that if an ulcer occurs on the anterior surface of the stomach, surgical interference is warranted in order to prevent the catastrophe of perforation which so frequently occurs when the anterior surface of the stomach is affected by ulceration.

In conclusion, the writer would express his thanks to those physicians and surgeons, and especially those on the staff of the London Hospital, who have allowed him to make use of their cases.

STATISTICS OF OPERATIONS IN PERFORATED GASTRIC ULCER, COLLECTED FROM THE JOURNALS.

It will be noticed that some of the cases are incomplete, but the writer felt that it was necessary to put down all cases, either complete or incomplete, as the latter class of cases form a large proportion of the fatal cases.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
1 Female	20	...	12 hours	Anterior surface near pylorus	Irrigation and suprapubic drainage	Recovery	...	B. M. J., 1898. Vol. I., p. 815
2 Female	Young	Twelve months previous dyspepsia and hæmatemesis	2½ hours	...	Irrigation	Recovery	...	B. M. J., 1898. Vol. I., p. 818
3	4 days	Death	Purulent peritonitis	B. M. J., 1898. Vol. I., p. 1656
4	Posterior surface	...	Death in 1 week	Perforation not found	Do.
5	Dyspepsia for two years	5½ hours	Anterior surface near cardia	Sponging, irrigation, drainage	Death in 1 week	Subphrenic abscess	B. M. J., 1898. Vol. II., p. 84
6 Female	35	...	2½ hours	Lesser curvature	Drainage	Recovery	...	B. M. J., 1898. Vol. II., p. 150

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
7 Female	20	...	10½ hours	Posterior surface near cardia	...	Death	? cause. 3vi. of blood brought up just before death	B. M. J., 1898. Vol. II., p. 1687
8 Female	13	Recovery	...	Do.
9 Female	18	Operation for adhesion due to perforation	...	Recovery	...	Do.
10	3 hours	...	No drainage	Recovery	...	B. M. J., 1899. Vol. I., p. 87
11	14 hours	...	No drainage	Death in 14 days	Subphrenic & perisplenic abscess	Do.
12	Acid eructation and slight vomiting two days before perforation	12½ hours	Anterior surface near cardia	...	Recovery	...	B. M. J., 1899. Vol. I., p. 475
13 Male	72	Sponged	Recovery	...	B. M. J., 1899. Vol. I., p. 962?

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
14 Female	21	...	13 hours	Anterior surface near cardia	...	Death	Subphrenic abscess	...
15 Female	19	Dyspepsia and anemia for two days	3 hours	Recovery	...	B. M. J., 1899. Vol. I., p. 962
16 Female	21	...	4½ hours	Anterior surface near cardia	Irrigation, with boiled water at 105°	Recovery complicated by left empyema	...	B. M. J., 1897. Vol. I., p. 389
17 Female	22	...	24 hours	Anterior surface near cardia	...	Death
18 Female	21	...	60 hours	Anterior surface near cardia	Suprapubic drainage	Recovery	...	B. M. J., 1897. Vol. I., p. 914
19	24 hours	Lesser curvature	...	Recovery	...	Do.
20 Female	Young	Dyspepsia; pain after food	12 hours	Anterior surface	Drainage	Recovery	...	B. M. J., 1897. Vol. II., p. 454
21 Female	Young	...	? 4 days	Posterior surface near cardia	...	Death	Ulcer not found, subphrenic abscess	Do.

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
22 ...	25	Greater curvature, extending on to anterior and posterior surface	Gastric fistula, separated by tampons	Death in 8 weeks	Ulcer still progressing	B. M. J., 1896, Vol. II, Epitome 53
23 Female	18	...	9 hours	Anterior surface nearer pylorus than cardia	...	Recovery	...	B. M. J., 1896, Vol. II, p. 1324
24 Female	18	...	6 hours	Anterior surface	Sponged	Recovery	...	Do.
25 Female	17	Indigestion and vomiting of food primarily. Severe gastric pain for a week	Statim	Anterior surface nearer pylorus than cardia	Sponged. No irrigation	Recovery	...	B. M. J., Vol. I., 1896, Epitome 399
26	Considerable time	Death	...	Do.
27 Female	22	Symptoms of gastric ulcer with hæmatemesis three years before	48 hours	Recovery	...	B. M. J., 1896, Vol. I., p. 1442

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
28 Female	Young	Dyspepsia for 2 years	10 hours	Anterior surface	...	Recovery	...	B. M. J., 1895. Vol. I., p. 198
29 Female	31	Symptoms of gastric ulcer for years	9 hours	Anterior surface	...	Recovery. Ulcer not closed up. Paul's tube used. Gastric fistula closed in 2 weeks after removal of tube	...	B. M. J., 1895. Vol. I., p. 759
30	None	...	Anterior surface. Adhesion had formed	Sponged with weak perchloride of mercury	Death in 15 days	Subphrenic abscess and septicæmia	B. M. J., 1895. Vol. I., p. 1093
31 Female	24	Recovery	Perforation was not found	B. M. J.
32 Female	...	Dyspepsia for 2 years—severe epigastric pain and vomiting for some months previously	7½ hours	Anterior surface	Irrigated and sponged	Recovery	...	B. M. J., Vol. II. 1895, p. 14

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
33 Female	20	Indigestion for 3 years	26 hours	Anterior surface	Irrigated and sponged	Death 2 weeks after	Subphrenic abscess and double pneumonia with abscesses in lung	B. M. J., 1895, Vol. II., p. 78
34 Female	20	Previous dyspepsia	5 hours	Anterior surface near cardia	...	Recovery	...	B. M. J., 1894, Vol. I., p. 576
35 Female	23	...	12 hours	Death	Imperfect cleansing	Do.
36 Female	14	Pain after food for some time	9 hours	...	Irrigation	Recovery	...	Do.
37 Female	20	...	4 hours	Death	Imperfect cleansing at the back of the stomach	Do.
38	Previous chronic constipation	26 hours	Duodenum	Irrigation. Suprapubic drainage	Death	General peritonitis	B. M. J., 1894, Vol. II., p. 859

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
39 Female	21	Gastric symptoms for some years	Not within 36 hours	Anterior surface near cardia	...	Death in 24 hours	Imperfect closure of perforation; renewed fouling leading to peritonitis	B. M. J., 1894. Vol. II., p. 859
40	Anterior and posterior surfaces	...	Death 36 hours after operation	Second perforation on posterior surface after that in anterior surface had been sewn up	Do.
41 Female	24	Epigastric pain for 6 months previous	4 hours	Anterior surface close to pylorus	Irrigation	Recovery
42 Female	26	...	14 hours	Anterior surface	Gastric fistula	Death in 6 weeks	Subphrenic abscess	B. M. J., 1893. Vol. I., p. 952
43 Female	22	None	6-9 hours	Anterior surface	Irrigation and drainage	Death in 24 hours	Ulcer not found, general peritonitis	B. M. J., 1893. Vol. I., p. 1258

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
44 Female	29	Previous suspicion of gastric ulcer	20 days	Parasplenic abscess due to perforation, opened and drained	...	Recovery	...	B. M. J., 1893. Vol. I., p. 1258
45 Female	20	Three years before had been treated for gastric ulcer	4 days	Anterior surface	Irrigation	Death	General peritonitis	Do.
46 Female	17	Three weeks' vomiting	3½ hours	Lesser curvature near cardiac end	Irrigation	Death in 45 hours	General peritonitis	B. M. J., 1893. Vol. II., p. 1044
47	2 hours	Posterior surface	Irrigation, suprapubic drainage	Death on 9th day	Peritonitis limited to pelvis	B. M. J., 1893. Vol. II., p. 864
48 } 49 }	24 hours	Anterior surface	Gastric fistula	Dead within 6 hours	...	B. M. J., 1894. Vol. II., p. 866
50 Female	32	...	3 hours	Anterior surface near cardia	Irrigation and suprapubic drainage	Recovery	...	B. M. J., 1894. Vol. II., p. 982 and 1426
51 Female	22	...	Under 12 hours	Anterior surface near cardia and pylorus	Suprapubic drainage	Death	Perforation not found	B. M. J., 1892. Vol. I. p. 63

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of Death.	Reference.
52 Female	20	History of indigestion for some years	4 days	Anterior surface	Sponged and irrigated	Death 30 hours after operation	General peritonitis	Do.
53 Female	23	Three years' symptoms pointing to gastric trouble	3 weeks	...	Drainage through left intercostal space	Death	Subphrenic abscess	B.M.J., Vol. I., epitome, p. 45
54 Female	24	Chlorosis	...	Anterior surface near cardiac end	...	Death in 1 week	Subphrenic abscess, pneumothorax	Do.
55 Female	24	Symptoms of gastric ulcer 1 year before	Drainage	Recovery	...	Do.
56 Female	23	Acute gastritis 2 years before, followed by gastric trouble since	Not within 2 days	Anterior surface	Drainage	Death	Abscess in front of stomach and transverse colon	Do.
57 Male	46	Previous gastric ulcer for 4 years	Death	...	B.M.J. 1891. Vol. I., supplement, p. 2

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
58	Posterior surface	...	Recovery	...	Lancet, 1899. Vol. I., p. 95
59 Female	4 hours	Anterior surface	...	Recovery
60 Female	27	Previous dyspepsia and hæmatemesis some months before	8 hours	Anterior surface nearer cardia than pylorus	Sponged carefully, drainage	Recovery	...	Lancet, 1899. Vol. I., p. 372
61 Female	...	None ...	24 hours	...	Sponged and washed	Death in few hours	General peritonitis	Do.
62 Female	20	Pain recurring every 3 months in stomach	33 hours	Anterior surface	Irrigation	Recovery retarded by perforation giving way and formation of subphrenic abscess	...	Lancet, 1899. Vol. I., p. 852
63 Female	17	Previous dyspepsia and hæmatemesis	13½ hours	Anterior surface	Irrigation and drying	Recovery	...	Lancet, 1899. Vol. I., p. 897

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
64 Female	Middle age	...	6 hours	Posterior surface	...	Recovery	...	Lancet, 1899. Vol. I., p. 902
65 Female	21	Previous history for some months of gastric ulcer	Not within 12 hours	Anterior surface	Sponged and irrigation drainage	Recovery	...	Lancet, 1898. Vol. I., p. 406
66 Female	16	Previous history of gastric ulcer for 18 months	2 days	Anterior surface	Sponged, not irrigated	Recovery	...	
67 Female	26	Several years' history of dyspepsia and vomiting but no hæmatemesis	72 hours	Anterior surface Near cardia	Sponged, not irrigated	Recovery	...	
68 Female	30	Some years' history of dyspepsia and hæmatemesis, æt. 19	48 hours	...	Sponged	Recovery	...	
69	3 hours	Anterior surface	Suprapubic drainage	Recovery
70	2½ hours	Anterior surface near cardia	...	Recovery	...	Lancet, 1898, Vol. I., p. 1323

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
71 Female	33	Previous history of dyspepsia, vomiting and hæmatemesis	4½ hours	Anterior surface near pylorus	Intestine turned out and sponged	Recovery	...	Lancet, 1898, Vol. I., p. 1323
72 Female	35	Lesser curvature one inch from the pylorus	Sponged	Recovery
73	26 hours	Death	...	Lancet, 1898, Vol. II.
74	18 hours	Recovery	...	Do.
75 Female	37	Previous pain and vomiting which has no relation to food	5 hours	Ulcer not closed up	...	Death	Inability to retain food, which passed out through fistula.	Lancet, 1898, Vol. II., p. 1761
76 Female	22	Anæmia and pain after food for 5 years	24 hours	Anterior surface	Irrigation	Recovery
77 Female	24	Pain after food and nausea	6½ hours	Anterior surface near pylorus	Irrigation and suprapubic drainage	Recovery

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS--continued.

Sex.	Age.	Previous symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
78 Female	19	Hæmatemesis two years previously.	6 hours	Anterior surface near cardia	Irrigation, sponging drainage	Recovery
79 Female	25	...	24 hours	Anterior surface	Irrigation, drainage	Recovery
80 Female	16	...	21 hours	Posterior surface	Irrigation	Death in 15 days	Subphrenic abscess	...
81 Female	19	...	32 hours	Recovery
82 Male	45	Five years' dyspepsia, emaciation, dilated stomach	6 hours	Anterior surface near pylorus	Irrigation and sponging, drainage	Death in 15 days	Pneumonia	Lancet, 1896, Vol. II., p. 11
83 Male	41	None	24 hours	Anterior surface nearer pylorus than cardia	...	Death	...	Do.
84 Male	32	Recovery	...	Do.
85 Female	20	...	8½ hours	Anterior surface near cardia	Sponged, drainage	Recovery	...	Lancet, 1896, Vol. II., p. 1584

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous Symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
86 Female	24	...	7½ hours	...	Sponged, drainage	Recovery	...	Lancet, 1896, Vol. II., p. 1584
87 Female	27	...	32 hours	...	Sponged, drainage	Recovery	...	Do.
88 Female	23	Death in 15 days	Subphrenic abscess	Do.
89 Female	23	Epigastric pain after food for some years	28 hours	...	Sponged, drainage	Death	Do.
90 Female	17 hours	...	Drainage	Death in 12 hours	...	Do.
91 Female	20	...	3½ hours	Death in 36 hours	General peritonitis	Do.
92 Female	28	Gastric trouble for some years, hæmatemesis once or twice	6 hours	Anterior surface near cardia	Irrigation	Recovery
93 Female	26	Hæmatemesis 3 years before and dyspepsia several years	6½ hours	...	Irrigation, no drainage

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous Symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
94 Female	19	Left-sided pain after food for 3 months	3½ hours	Anterior surface near cardia	...	Recovery
95 Female	38	Treated for gastric ulcer 6 months previously	7½ hours	Posterior surface close to lesser curvature	Sponging and irrigation	Recovery	...	Lancet, 1900. January 20
96 Female	19	Anorexia and pain after food for some time	9 hours	Anterior surface	...	Recovery	...	Lancet, 1895. Vol. II. October 19
97 Female	...	Ten years previously had suffered from severe symptoms of gastric ulcer	19½ hours	Lesser curvature	Irrigation, sponging.	Death in 13 days	Local abscess between small intestine and abdominal wall	Do.
98 Female	17	None	Under 24 hours	Anterior surface near cardia	Irrigation	Death in 3 days	Second perforation on posterior surface	Lancet, 1895. Vol. II.
99 Female	21	Dyspepsia for previous six months. Pain after food relieved by vomiting. No hæmatemesis	19½ hours	Anterior surface	Irrigation, drainage.	Death	Second perforation on posterior surface	Lancet, 1895. Vol. II., p. 264

STATISTICS OF OPERATIONS, COLLECTED FROM THE JOURNALS—continued.

Sex.	Age.	Previous Symptoms.	Time elapsing between perforation and operation.	Position of perforation.	Methods of cleansing peritoneum.	Result.	Cause of death.	Reference.
100 Female	...	Three years symptoms of gastric ulcer	Within 24 hours	Death	General peritonitis	Lancet, 1894, Vol. I., p. 413
101 Female	20	Gastrodynia and vomiting after food for nearly four years	17½ hours	Anterior surface pain referred to left shoulder	Sponged	Recovery
102 Female	...	Previous history of gastric ulcer for two years	24 hours	Posterior surface near cardia	...	Recovery
103 Female	26	Under 24 hours	Posterior surface near cardia	...	Recovery	...	B. M. J., 1892

CASES OF PERFORATION COLLECTED AT GUY'S HOSPITAL, 1885-1898.

	Sex.	Age.	Position of Perforation.
1	Male ...	46	Anterior surface; four inches from cardia, one and a half from lesser curvature.
2	Male ...	40	Anterior surface; one inch from pylorus.
3	Female	Anterior surface; near pylorus.
4	Female ...	38	Posterior surface; two inches from cardia.
5	Female ...	34	Gastrocolic fistula.
6	Male ...	57	Anterior surface; near pylorus and lesser curvature.
7	Male ...	45	Anterior surface; near pylorus.
8	Female ...	36	Anterior surface; near greater curvature.
9	Female ...	19	Anterior surface.
10	Male ...	60	Anterior surface.
11	Male ...	67	Anterior surface; near lesser curvature and cardia
12	Female ...	36	Anterior surface.
13	Male ...	40	Anterior surface; two and a half inches from cardia.
14	Female ...	28	Anterior surface; near lesser curvature.

CASES OF PERFORATION COLLECTED AT GUY'S HOSPITAL, 1885-1898—continued.

	Sex.	Age.	Position of Perforation.
15	Female	20	Anterior surface.
16	Male	37	Anterior and posterior surfaces.
17	Male	40	Anterior surface; near lesser curvature and cardia.
18	Female	41	Lesser curvature; two inches from pylorus.
19	Female	61	
20	Female	22	Anterior surface; cardiac end.
21	Female	13½	Anterior surface; two inches from lesser curvature.
22	Female	28	Anterior surface; four inches from pylorus.
23	Female	17	Anterior surface.
24	Male	19	Lesser curvature.
25	Female	25	Lesser curvature; four inches from pylorus.

CASES OF HÆMATEMESIS BRIEFLY TABULATED SO AS TO SHOW
THE SOURCE OF THE HÆMORRHAGE.

- 1.—Indurated ulcer in first and second parts of duodenum on posterior wall ; base formed by pancreas ; orifice in aneurism of artery crossing ulcer.
- 2.—Ulcer situated just beyond pylorus on posterior wall ; base formed by pancreas ; no obvious source of bleeding.
- 3.—Ulcer situated just beyond pylorus on posterior wall ; orifice in large artery passing through head of pancreas.

In all these cases death was due to acute hæmorrhage.

- 4.—Ulcer situated just beyond pylorus on anterior wall, giving rise to recurrent hæmatemesis, exhaustion and death.

The above cases are recorded by Kelynack, Brit. Med. Journ., 27th October, 1898.

- 5.—Ulcer situated on posterior surface of stomach, thrombosed vessels radiating therefrom ; recurrent hæmatemesis ; collapse, operation, death.—Tubby, Lancet, vol. i., 1899, p. 1267.
- 6.—Gastric ulcer ; recurrent hæmatemesis for four years ; exhaustion, operation, death.—Mr. Openshaw, London Hospital Reports.
- 7 and 8.—Ulcers situated at pylorus on posterior surface, leading to severe recurrent hæmatemesis.—Mr. Mayo Robson.
- 9.—Ulcer situated near pylorus on posterior wall, adherent to pancreas ; recurrent frequent hæmatemesis.—Küster, Centralblatt, für Chirurgie, 1894, No. 30.
- 10.—Small linear ulcer close to cardiac end ; hæmatemesis, melæna, exhaustion and death ; post-mortem, stomach and intestines filled with blood. Monro, Lancet, vol. i., 1896, p. 994.
- 11.—Ulcer situated two inches from pylorus on the lesser curvature ; large size ; severe hæmatemesis ; rapid death, bowels full of blood.—Mr. Mayo Robson, Leeds Infirmary Reports.
- 12.—Mr. Mansell Moullin's case recorded above.

The following cases are from the Guy's records.

- 13.—W. D., æt. 42, heavy drinker. Hæmatemesis ; death occurring rapidly ; submucous hæmorrhages found at cardiac end.
Post-mortem Reports, 1886, No. 436.
- 14.—A. C., æt. 24, admitted under Dr. Perry, 22nd July, 1896, for hæmatemesis death. Ulcer found half-way between pylorus and cardia on lesser curvature ; crossing floor there was a small artery in which there was a perforation.
- 15.—Male, æt. 40, circ., admitted under Mr. Symonds, October, 1899. Subacute hæmorrhage ; death ; ulcer half an inch beyond pylorus occupied by aneurism of artery in which there was a small hole.
- 16.—Female, æt. 35, admitted 31st July, 1891. Recurrent hæmatemesis, melæna and death ; ulcer with cicatricial edges found at pylorus on posterior surface.

- 17.—Male, æt. 35. Ulcer situated on posterior surface of stomach, exposing pancreas, leading to recurrent frequent hæmatemesis, exhaustion and death.

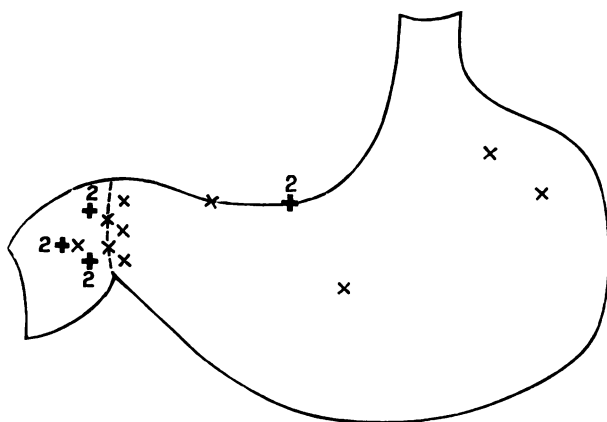


Diagram illustrating source of bleeding in ulcer of stomach.

Crosses marked 2 indicate arterial origin.

Other crosses indicate origin from raw surface of ulcer.

POISONING BY LEAD CHROMATE.

By T. WILSON SMITH, M.D.

IN the latter part of the summer of 1893, several cases of lead-poisoning of a rather severe type, due to the consumption of home-made wines, were admitted into the Royal United Hospital, Bath, under my care, in the absence of Dr. Fox, late senior physician.

My attention was drawn to home-made wine as a source of lead-poisoning by a note in the *Lancet*, 1886, upon the Report of Dr. Campbell, medical officer of health for Calne, on an outbreak of cases of lead-poisoning.

"The cases occurred chiefly in men . . . and coincided roughly with harvest work. Examinations of drinking water, beer, tea and coffee, bread, and various cooking utensils gave no light.

"The occurrence of seven acute cases . . . two almost fatal, led to the discovery of the mischief.

"The wife of one of the men explained that her husband had drunk some home-made rhubarb wine. She was sure 'it could not contain poison of any sort, as she was very careful in selecting and cleaning her rhubarb . . . besides the vessel was a beautifully clean glazed earthenware pan in which she steeped it for a fortnight or three weeks to ferment.'"

Dr. Campbell says: "As is well known this glaze contains sixty per cent. of white or red lead, and during the process of fermentation the acids of the fruit used, with the acetic acid generated, act on the lead, forming acetate of lead.

“Not only rhubarb, but red and black currants, sloes, and damsons are so used, and some thrifty wives have been able to supply their husbands with gallons for use in harvest time.”

Dr. Campbell recommended that the wine should be made in wooden vessels.

The foregoing cases in the hospital gave the ordinary signs of plumbism, and presented no difficulty in diagnosis; but the following case, which was due to poisoning by plumbic chromate, taken in home-made wine, was for some time sufficiently puzzling, the more so as by some slip the lead-line on the gums was not at first detected.

John C., æt. 31, a grocer, of alcoholic history, living at a village some miles out of Bath, was admitted into the Royal United Hospital, August 15th, 1893, having been sent in as a case of intestinal obstruction for laparotomy. Dulness was said to be marked in the right iliac fossa, and a hard mass to be felt towards the right side.

His history was that ten days previously he was taken ill with pains in the loins and bowels. There had been absolute constipation for five days before admission, not even flatus passing. He had severe retching the day that constipation commenced, and had had vomiting from that time till admission.

On admission, pain was noted to be general over the lower part of the abdomen, but especially severe in the right iliac fossa. The abdominal walls were lax, and there was no tympanitis. Laparotomy was not thought necessary, and he improved under treatment; but as he relapsed on August 28th, pain and vomiting recurring, and his sallow colour became deeper yellow, the conjunctivæ also becoming icteric, he was transferred to a medical ward under my care. The urine, which was of a dark colour, resembling that containing bile, was repeatedly tested at intervals for bile, but, notwithstanding the resemblance, no bile was ever found, either by the iodine or nitric acid tests. The bowels were opened by enemata, and he again improved. But on September 6th, pain and vomiting came on again, and continued, with obstinate constipation. Nutrient injections were given but soon returned, and the patient's condition became serious.

On again examining the mouth I found a well-marked lead-line, beside pigmentation of the mucous membrane lining the cheeks; and the fur having cleared away, the tongue showed pigmentation of the papillæ of a bluish-black colour along the left margin of the dorsum, resembling somewhat the appearance of an ill-shaven chin in a person of dark complexion.

Appropriate treatment for the plumbism was soon followed by relief and recovery, but the apparent jaundice still remained obscure in causation until I found a reference to a series of cases of poisoning by lead chromate reported by Dr. R. Smith, of Ardwick, in the *Lancet* of 1882.

Dr. R. Smith mentions three typical cases, two of whom suffered from yellow discolouration of the skin and conjunctivæ, sometimes intense; the urine occasionally contained albumen, with a trace of chromium.

The patients recovered with difficulty, the symptoms consisting generally of obstinate vomiting, constipation or purging with sap-green motions, and apparent jaundice. Over sixty cases occurred, all in connection with one cloth mill, where in weaving an orange-coloured cloth a yellow dust was given off.

A fatal case occurring led to an inquest, and more stringent regulations were followed by the cessation of the disease.

Microscopic examination of the fibres of the cloth made from the yarn showed minute crystals of orange-brown (chromate of lead).

After reading these cases I came to the conclusion that John C. had derived his lead-poisoning from plumbic chromate in the glaze of the fermenting pan in which the home-made wine was manufactured. On enquiry, however, at the local pottery in Bath, it transpired that litharge, or red oxide of lead, is the ingredient used in glazing the earthenware pans commonly sold for fermenting purposes.

John C. must therefore have obtained his fermenting pan elsewhere, and I was informed that yellow chrome (lead chromate) is an ingredient of the glaze used in some pottery works in other localities in the manufacture of earthenware vessels.

On referring to Miller's Chemistry, and other works on Chemistry also, the statement is found that lead chromate is insoluble in water, or acids, though soluble in excess of soda or potash.

At first sight this might seem to negative the possibility of the acids, generated by fermentation, acting on a glaze containing lead chromate ; but Mr. Gatehouse, analyst for the City of Bath, informs me that this statement is a relative one, and in some experiments, conducted at my request, he succeeded in dissolving, after some hours, a portion of the orange chrome in a mixture of acetic and malic acids, one per cent. of each ; and this percentage is one which obtains in fermentation of fruit.

I never succeeded in obtaining a piece of John C's. fermenting pan for analysis, though it was promised to me.

Some months later he was practically well, all pigmentation having disappeared from the tongue and the mouth with the exception of a lead-line on the gums, which still persisted. On attempting to trace him this year, 1899, I found that he had left the locality.

His case is interesting from the pigmentation of the dorsum of the tongue, hitherto not recorded in cases of lead poisoning, so far as I am aware. Dr. Oliver does not describe it in his monograph, or in Clifford Allbut's System of Medicine, though of course the staining of the mucous membrane of the cheeks has long been observed.

Cases of plumbic chromate poisoning, should a lead-line on the gums be overlooked, may be mistaken for various diseases attended with jaundice and abdominal pain, but though the urine looks as if it contained bile, testing will show its absence.

That poisoning with plumbic chromate is rare may be gathered by the absence of any description of its typical symptoms except in the paper of Dr. R. Smith, of Ardwick, previously referred to. It is perhaps remarkable that in none of the cases recorded have ulceration or perforation of the nasal septum occurred, thus contrasting with the cases of potassic chromate poisoning frequently met with in French polishers, in which perforation is common.

ON THE PRODUCTION OF GAS-CONTAINING CAVITIES IN THE INTERNAL ORGANS OF THE BODY.

By J. H. BRYANT, M.D.,

AND

W. C. C. PAKES.

WE have long felt that the formation of Gas-containing Cavities in the Internal Organs was an ante-mortem rather than a post-mortem change. For some time we were on the look out for a suitable case to investigate thoroughly from every point of view. Such a case as we fully report below came under our notice in February, 1897, and, as will be seen, there is very good evidence in this case at least in favour of the ante-mortem theory. In addition to satisfying ourselves on this point we have worked out, as far as possible, the pathology of the condition as illustrated by our own case and by similar cases which have been published.

CASE 1.—E. R., female, æt. 51. Admitted 1st February, 1897, for abdominal pain, under the care of Dr. Pitt, who has kindly allowed us to use the clinical notes of the case. She has had fifteen children, has always enjoyed good health, and has been temperate in her habits. Fourteen days before admission she was feeling quite well until she was suddenly attacked with severe pain in the abdomen, followed by jaundice. Her tongue became very dry: she was obliged to take to her bed and have warm flannels applied to her abdomen. Her bowels have been

regular and the motions were normal in appearance. For the last three days she has been passing porter-coloured urine and micturition has caused much pain.

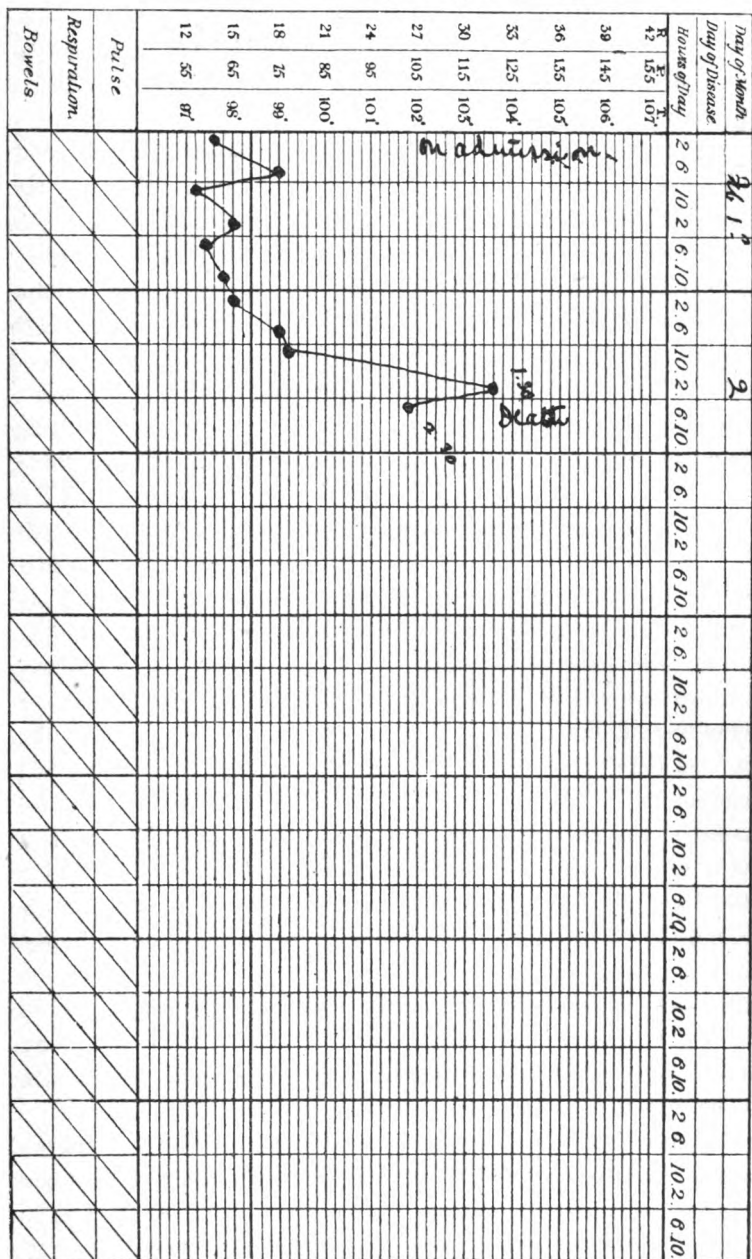
Condition on admission.—Pulse 80, respiration 40, temperature 97·6°. She was brought up to the hospital in an ambulance, and appeared to be suffering from severe pain in her abdomen. Her breathing was extremely rapid. She was an enormously fat woman, and was jaundiced. Her tongue was very dry and she felt extremely thirsty. She was quite unable to move and was groaning with the pain in her abdomen. Her liver was thought to be enlarged, but it was impossible to make a satisfactory examination. There was no ascites. No cardiac impulse could be seen and the sounds were feeble and indistinct, suggesting fatty degeneration and infiltration of the myocardium. There was slight bronchitis.

Urine 1010, acid; a trace of albumen present. No sugar, blood, or bile found.

Progress and treatment.—She was put to bed at once and placed on her right side, this being the most comfortable position for her. At 12 p.m. the abdominal pain was less.

February 2nd. She slept the greater part of the night and said she felt better. The abdomen was found to be quite supple, but was still tender on palpation. An enema was given, but it was partly returned without bringing away any fæces. She was seen by Dr. Pitt in the afternoon and he diagnosed gall-stones. Later in the afternoon she passed a large quantity of urine. At 10 p.m. she was sleeping comfortably; temperature 99·2°, pulse 92, respiration 86. At 1.30 a.m., February 3rd, temperature 103·6°, pulse 116, respiration 68. The heart action was very irregular, and she began to get blue. At 2.30 a.m., temperature 101·8°. The cyanosis had increased, the breathing became slower and gasping in character, and she died at 3.15 a.m.

Autopsy.—Eleven hours after death (aerial temperature 10° C.) rigor mortis was well marked, and there was no evidence of post-mortem decomposition. Brain, forty-seven ounces, healthy. No pleurisy. The bronchial tubes were full of muco-pus. About one and a half ounces of blood-stained fluid were found in the pericardial



sac. Numerous little thin transparent blebs containing gas projected from the surface of the pericardium (visceral) and gave a sensation of crackling like surgical emphysema on rubbing the finger over them. The right ventricle was invested with a thick layer of fat, and its wall which was infiltrated with fat appeared to be in a condition of fatty degeneration. The muscle of the left ventricle was also pale and appeared to have undergone fatty degeneration and infiltration. The endocardium lining the cavities and covering the valves was deeply stained with blood. The intima of all the vessels was also deeply stained with blood. The liver weighed seventy-five ounces; it was a little lighter in colour than normal, was very soft, and on being handled gave rise to a sensation of emphysema-like crackling. On section it was found to be very soft, and a large number of small cavities and cysts containing gas were found. The gall-bladder was soft and friable and contained a number of faceted gall-stones and muco-pus. The common duct was dilated and full of calculi, the lower end being blocked by a large calculus about two inches in circumference. The spleen weighed sixteen ounces; it was very soft, and on palpation gave a sensation of emphysema-like crackling similar to that in the liver. On section it was found to be soft, and a large number of small cavities containing gas were seen. The kidneys weighed nineteen ounces; the capsules peeled off with ease. Both organs were very soft, especially the right, in fact so soft that when held in the hand, the long axes being at right angles to the palm of the hand, they no longer retained their reniform shape but collapsed into a globular form. On pressing them emphysematous crackling could be obtained. On section it was difficult to distinguish cortex from medulla: they were soft and friable, numerous small cysts containing gas were found, and both appeared to be in a state of advanced decomposition.

Sections of the liver prepared for microscopical examination and stained with gentian violet showed a large number of the small cavities which contained gas, the largest about one-third of an inch in diameter, the smallest being only perceptible under the microscope. Many of the cavities were circular, some were oval,

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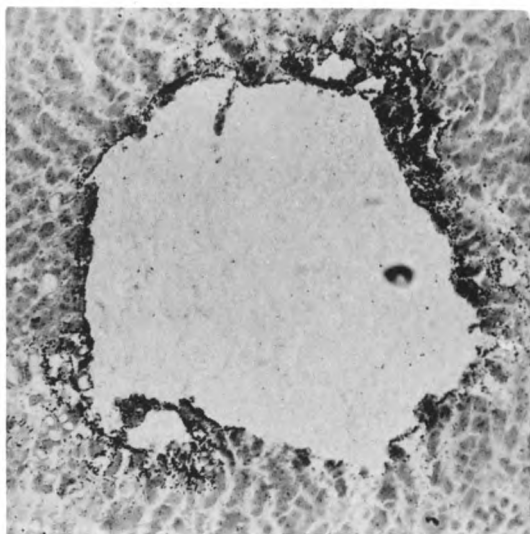


FIG. 1.—Section of Liver showing a gas-containing cyst surrounded by colonies of Bacilli. $\times 60$.

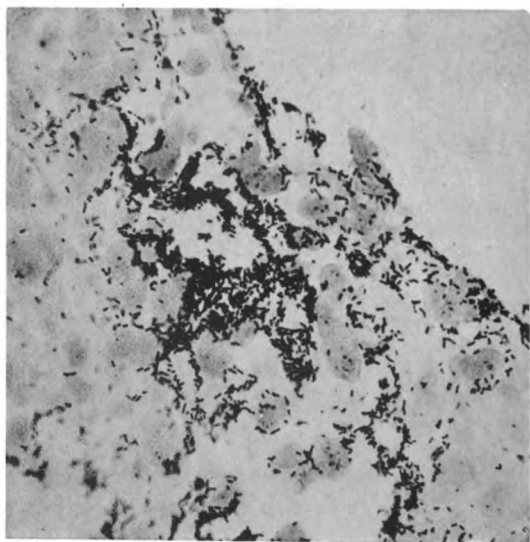


FIG. 2.—Section of Liver showing a part of the edge of a cyst. $\times 250$.

and others irregular in form, but the majority had quite smooth and regular edges. No definite lining membrane could be detected in any way resembling the intima of a dilated blood-vessel or the epithelial lining of a bile-duct. There was no increase of connective tissue and no collection of inflammatory cells bordering the cysts: they appeared to be in the parenchyma of the organ, and liver cells were found in all cases bordering them. These bordering cells seemed to be more closely packed together and more elongated than those in a normal lobule, suggesting not only actual destruction but also compression by the gas. Close up to the edge of the cavities and also lying just inside them a large number of short irregular bacilli stained a deep violet colour was seen forming distinct and continuous wreaths; these organisms, however, were not in a uniformly thick layer, for in places they were arranged together in distinct clumps; a few single bacilli and also large masses of them were seen lying free in some of the spaces. In addition small clumps of bacilli were found with commencing cavities in their centres in the parenchyma of the organ, which did not appear to have any definite relation to either portal veins, or hepatic veins, or bile-ducts. A few columns of organisms, however, were found as if lying in vessels cut in their longitudinal axes. It could be definitely made out that there was no general distribution of the bacilli between the liver cells throughout the section; they were distinctly arranged in groups as described above. The liver cells did not stain readily nor did their nuclei, and they had a very cloudy and finely granular appearance; a few showed the presence of hæmatin granules and some had undergone fatty degeneration.

A section of the spleen showed a general dissemination of the bacilli throughout. They did not appear to be confined to the periphery of the gas-containing spaces as in the liver, or to be arranged in clumps.

The bacilli were short with rounded ends, varying in length from one and a half to three times their breadth. Many were joined together and appeared like elongated diplococci; a few were considerably longer than the majority, being almost five

times as long as broad. By no method of staining could capsules be found.

Bacilli in the sections subjected to Gram's method did not retain the stain.

Bacteriological examination.—The spleen contained a large number of bacilli, many of which were in the form of diplobacilli. None of these retained the stain after treatment by Gram's method. Many of them had what appeared to be a slightly developed capsule.

Cultivations made from the spleen resulted in a pure culture of the bacillus coli communis. Anaerobic cultivations produced only this bacillus. There was no sign of the bacillus aerogenes capsulatus.

The bacillus thus obtained produced abundant indol in broth after forty-eight hours' growth. It clotted milk in twenty-four hours with an acid reaction. On agar streak-cultivations an opalescent moist growth was seen. Gelatin was not liquefied, but a spreading flat growth was produced with crenate edges. The colonies on gelatin plates were quite typical. Abundant gas was produced in gelatin shake-cultures. Young agar-cultures gave fairly motile bacilli possessing from four to eight flagella when stained by Pitfield's or McCrorie's method.

The organism possessed a marked virulence.

1. A guinea-pig weighing about 500 grams was inoculated intraperitoneally with 1 c.c. of a twenty-four hours old broth-culture. It was dead next day and typical colon bacilli were found in large numbers in the blood in the heart, the spleen, and peritoneal fluid.

2. A guinea-pig was inoculated intraperitoneally with .006 grams of a twenty-four hours old agar-cultivation, and died at the end of twenty hours. The bacillus coli communis was found in large numbers in the blood in the heart, the spleen, and peritoneal fluid.

3. A rabbit weighing about 1500 grams was inoculated intraperitoneally with 1 c.c. of a twenty-four hours old broth culture of the organism, and died the next day. The bacillus coli

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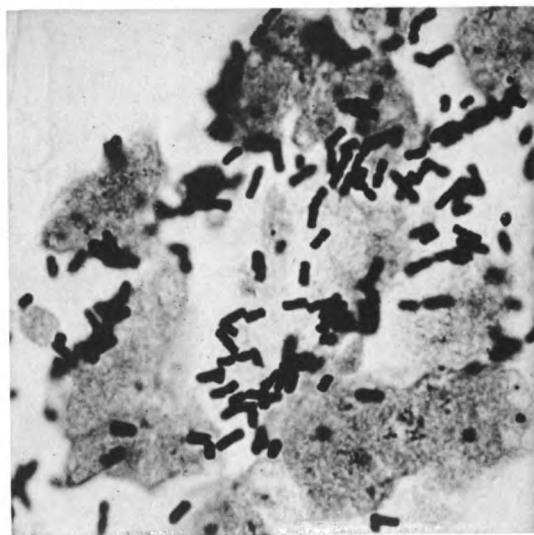


FIG. 3.—Section of Liver, similar to fig. 2, but more highly magnified.
× 1,000.

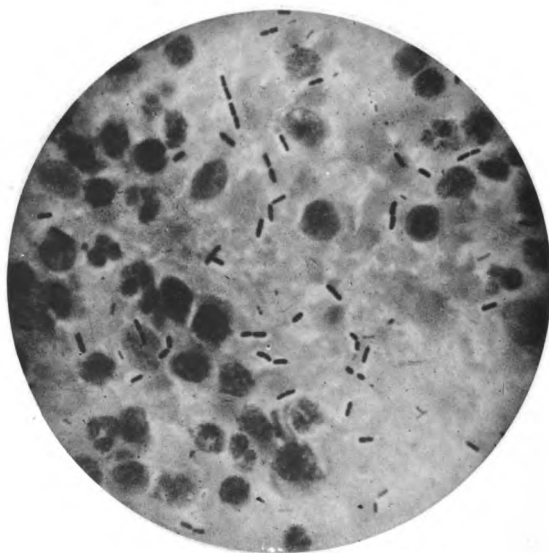


FIG. 4.—A Spleen Rub. × 600.

E. C. BONSFIELD, Fecit.

communis was found in the blood in the heart, the spleen and peritoneal fluid.

4. A rabbit weighing about 1600 grams was inoculated intraperitoneally with about .006 grams of a twenty-four hours old agar-culture. It died the next day, and the bacillus coli communis was found in the blood in the heart.

5. A guinea-pig weighing about 500 grams was inoculated subcutaneously with a bacillus coli communis obtained from normal fæces. An abscess containing bacillus coli communis was found at the seat of inoculation. The abscess was opened, and the animal got well. It was then inoculated intraperitoneally with .006 grams of a twenty-four hours old agar-culture of the organism obtained from the heart-blood of guinea-pig No. 2. On the following day it was very ill, but in three days it had recovered. About a fortnight later it was killed. No bacillus coli communis could be found in the blood in the heart, the spleen, or liver, but a few were found in the peritoneal fluid.

It will be seen from the above description of the histological appearances of the affected organs, and the results of the bacteriological examination, that our case was due to the bacillus coli communis. We have not been able to find a similar case amongst the literature on the subject which has been definitely proved to be due to this micro-organism alone.

I.—CASE IN WHICH THE BACILLUS COLI COMMUNIS WAS PROBABLY PRESENT.

CASE 2.—A somewhat similar case has been described by L. Heydenreich, but no bacteriological examination was made and the liver was the only organ affected.

A soldier suffering from typhoid fever died of cardiac failure in the third week of the disease. Clinically it was only a severe case of typhoid fever without complications. There were no signs pointing to any disease of the liver. At the autopsy, extensive ulceration of the lower part of the ileum was found, the mesenteric glands were swollen and the spleen was enlarged, the kidneys hyperæmic, and the lower lobes of the lungs oedematous and in a condition of hypostatic congestion.

The liver was only slightly enlarged; on section it resembled in appearance a section of black bread. Numerous small round or oval cavities, varying in size from a poppy-seed to a pea, were found scattered throughout its substance. The microscope showed still smaller cavities. The capsule was normal, the parenchyma was coffee-brown in colour, and no definite lobulation could be distinguished; the section was smooth, moist, shiny, and slightly hyperæmic; pieces of it floated in water. Microscopic examination showed that the cavities were usually surrounded by well-defined vessel

walls, some showed a thin, scarcely visible, membrane, whilst others were surrounded by connective tissue and partly by cells, and others by liver cells alone.

These former conditions the author explains by the supposition that the cavities originated in the capillaries, whose thin walls remained or burst, and that others without a membrane originated outside the vessel walls.

The liver walls were swollen, cloudy, slightly granular, with indistinct nuclei which were difficult to stain; some of the cells contained hamatin granules and others showed signs of fatty degeneration. Sections treated with gentian violet and picric acid showed the tissues stained a yellow colour and numerous short regular bacilli stained a dark violet, these latter were seen to be congregated in enormous numbers at the peripheries of the cavities, around which they formed a kind of wreath, generally complete but here and there massed together. In some parts of the section clumps of bacilli were seen in the tissues without corresponding cavities or with small commencing microscopic cavities in the centre or at the end of the masses. No cavities were found without bacilli around them. In places clumps of bacilli were seen actually in the cavities. All these micro-organisms resembled typhoid bacilli in form and size but not in their staining properties.

The fact that the bacillus stained so well, and the very free production of gas, led to the assumption that it was the *bacillus coli communis*, or a near ally derived from the intestinal canal.

It was thought that the condition was most unlikely to have been an ante-mortem change, partly on account of the absence of any sign of hyperplasia, proliferation of connective tissue, irritation or inflammation, and partly from the fact that no objective symptoms during life, such as pain, jaundice or evidence of portal obstruction had been noticed.

In favour of the change being a post-mortem one was the time after death of the autopsy, viz., twenty-eight to thirty hours, and the temperature of the room in which the corpse had been during this time, viz., 18° to 20° C.

In this case the liver was the only organ affected, and that was put down to the fact that the liver contains a considerable amount of glycogen and sugar and that the *bacillus coli communis* in particular has the property of producing gas from glucose.

Göbel describes two cases in which the *bacillus coli communis* was found but not in pure culture, as will be seen below.

II.—CASES IN WHICH THE *BACILLUS AEROGENES CAPSULATUS* WAS FOUND.

CASE 3.—The micro-organism which has most frequently been found associated with this condition is the *bacillus aerogenes capsulatus*, first described by Welch and Nuttall in 1892, and obtained from the emphysematous tissues and blood of a man who died of aneurysm of the aorta, which had perforated by a small opening through the anterior thoracic wall, and given rise to repeated external hæmorrhages. No subcutaneous emphysema was noticed before death. The autopsy was performed in cool weather, eight hours after death. There was no evidence of post-mortem decomposition, or of emphysema of the subcutaneous tissues of the body. The blood vessels and heart contained abundant gas. The blood was lake coloured, and the endocardium of the heart and the intima of the blood vessels were deeply stained. Gas was also found in the blood-stained serum in the pericardial sac and in the peritoneum. Gas bubbles were present in

the myocardium, liver, spleen and kidneys, and in the ante-mortem thrombus in the aneurysmal sac. The gas burned with a bluish, almost colourless flame, a slight detonation being heard at the time of ignition.

Frozen sections of the fresh liver showed the small cavities, visible to the naked eye, to be surrounded by liver cells, much disintegrated and in a state of fatty degeneration. Bacilli were present throughout the liver, but were by far most abundant in and near the small cavities.

Cultivations from the blood and tissues resulted in the growth of a strict anaerobic bacillus (*bacillus aerogenes capsulatus*). No aerobic organism grew.

CASE 4.—Graham, Steward, and Baldwin report the case of a married woman, æt. 35, who was perfectly well until fourteen hours before death, when she was seized with a severe chill, which lasted more than four hours, and was followed by a pain in the region of the uterus and ovaries, and by vomiting and purging. She was seen five hours after the onset, and was then found to be restless, and to be suffering from pain in the lower abdominal region, and with a slight flow of bright red blood from her uterus. She was conscious and talked up to three minutes before death, which was sudden. An hour after death the body was found to be emphysematous all over.

The autopsy was performed twenty-four hours after death. The uterus gave evidence of a recent abortion. The vessels, tissues and organs of the body were enormously distended with gas, which ignited with an explosive sound, and was determined by chemical examination to be hydrogen. Bacilli were obtained from the blood in very large numbers, and were identified as the *bacillus aerogenes capsulatus*. This case certainly shows the ante-mortem invasion of the micro-organism at least in the subcutaneous tissues. The uterine lesion would suggest this organ as the probable point of invasion.

CASE 5.—P. Ernst reports the case of a woman who developed putrid endometritis and septic peritonitis after the removal of a macerated four months fetus. No development of gas was noticed in the tissues during life. At the autopsy, three hours after death, the entire uterine wall crepitated. Coagula in the right iliac vein and conus pulmonalis contained gas. Gas was also found in the liver and myocardium.

CASE 6.—The liver was similarly affected in another case, the autopsy on which was made eighteen hours after death. The cause of death was peritonitis resulting from traumatic perforation of the intestine in a hernial sac. The patient died four hours after a hernio-laparotomy. Green discolouration of the liver, and other parts, indicated post-mortem decomposition. Gas was found in the liver, and gas blebs in the intestinal mucosa.

Microscopical examination of the liver showed a disappearance of the nuclei and degenerative changes around masses of bacilli. The bacillus found and cultivated was the *bacillus aerogenes capsulatus*.

Gœbel reports three cases, in all of which the *bacillus aerogenes capsulatus* was found.

CASE 7.—1. This was a case of pyelonephritis, secondary to papilloma of the bladder and abscess of the prostate and seminal vesicles. Gas blebs were found in the myocardium, liver, spleen, adrenals and stomach. Around many, but not all, of the blebs in the heart and liver, the cells were necrotic. Necroses were not present in the spleen around the gas holes.

The *bacillus aerogenes capsulatus*, streptococci, and the *bacillus coli communis* were found.

CASE 8.—2. A case of enlarged prostate, which had been catheterised. General arteriosclerosis, pulmonary emphysema, lobular pneumonia and embolism of branches of the pulmonary arteries were found, in addition to gas blebs beneath the mucous membrane of the urinary bladder. Necroses were absent from the walls of these blebs. The bacillus aerogenes capsulatus was found; no other micro-organisms were described.

CASE 9.—3. A case of pyæmia, following endo-phlebitis of the cutaneous veins of the left foot. Gas blebs were found beneath the gastric and intestinal mucosa, and necroses were present in the neighbourhood of the blebs. The bacillus aerogenes capsulatus, the staphylococcus pyogenes aureus, and the bacillus coli communis were found.

Gœbel's general conclusion was that the bacillus aerogenes capsulatus of Welch and Nuttall, and the bacillus emphysematosus of Fraenkel were identical.

CASE 10.—Williams (Welch and Flexner's paper). A case of pyonephrosis. At the autopsy thirty-one hours after death the pelvis, calices, and ureters of both kidneys were dilated and filled with pus. Many small gas-containing cavities were found in the liver and myocardium. (Schaumleber.)

Cultures of the bacillus aerogenes capsulatus were obtained from the liver.

CASE 11.—(Case 21, Welch and Flexner's paper.) A negro, æt. 51, who for eleven years whilst in the epileptic ward of Bay View Asylum, Baltimore, used to have one or two attacks daily. On June 3rd he was in his usual health. At 5 a.m., on June 4th, he was found unconscious in bed; bubbles of reddish froth were seen to be issuing from his mouth and nose. He died twenty minutes later. An hour after death the body was swollen and emphysematous. Six hours later it was still more swollen. The autopsy was performed twelve hours after death. There was no rigor mortis. The upper part of the body was very swollen, the upper lids were puffed and emphysematous. The tissues of the penis and scrotum were distended with gas. The abdomen was distended and tympanitic. Gas was found in the peritoneal cavity free from putrefactive odour. It burnt with a blue flame and detonated on ignition. The large veins contained gas and the cavities of the heart were distended with blood and gas. Gas bubbles in large numbers were found in the liver and spleen. On section of the kidneys blood mixed with gas exuded from the cut vessels. Gas could be also squeezed from the lungs, and it burnt with a blue flame. The bacillus aerogenes capsulatus was found.

CASE 12.—(Case 22, Welch and Flexner's paper.) A man who died in the third week of typhoid fever. The autopsy was performed eighteen hours after death. No post-mortem decomposition. No emphysema or tympanites at the time of death. At the post-mortem examination emphysema of the neck, chest, axillæ and scrotum was found. The heart and blood-vessels contained gas. The endocardium was stained red. The serous sacs contained gas and blood-stained serum. The mediastinal tissues were emphysematous. The liver and spleen contained small gas cavities but were less distended with gas than the kidneys. There was no perforation or peritonitis. There was typical typhoid ulceration of the intestine. The bacillus aerogenes capsulatus was seen on cover-glass preparations and was obtained by culture.

CASE 13.—(Case 23, Welch and Flexner's paper). A man who died in the second week of typhoid fever. On the day before his death marked tympanites was noticed. The autopsy was performed eight hours after death. There was no odour or discolouration indicating decomposition. There was no swelling of the body. There was some slight emphysematous crackling on the outer side of the forearm below the elbow, on the wrists, and along the outer side of the thigh. There was no gas in the heart, vessels, or any organ except the liver, which was large, pale, and everywhere perforated with small gas holes. The gall-bladder and bile-ducts contained gas. The walls of the gall-bladder and the surrounding connective tissue were emphysematous.

Microscopical examination showed extensive necrosis of the liver. There was no perforation of the intestine. The presence of the bacillus aerogenes capsulatus was shown by microscopical examination, culture and inoculation. In this case it was thought that the gas-forming bacilli invaded the liver by means of the bile-ducts.

CASE 14.—(Adami's case.) A. C., æt. 45, a lumberman. For six years he had suffered from dull aching pains in the loins. He was temperate, and had no other complaint. Four weeks prior to his admission, while drawing wood, he felt a soreness over his whole body. On the following day pain superseded the soreness, and it was so severe that he had to take to his bed. He suffered from chills, his urine became red coloured (bilirubin), and a week before his admission, on April the 9th, he became jaundiced.

Condition on admission. He complained of extreme weakness, but was in no pain. Temperature 102°, pulse 120, respiration 28. The abdomen was pendulous, and it was tender, especially over a small area three inches to the left and five inches above the umbilicus, where an indistinct mass could be felt, which was dull on percussion. There was tenderness in the left groin. The urine contained albumen and bilirubin. The fæces were liquid, and contained blood and pus cells. Soon after admission he vomited coffee-ground material, and the vomiting continued until his death, which occurred five days after his admission.

On the 14th he was in great pain, and was constantly coughing. The abdomen was enlarged and tympanitic, and the tympanitic note could be obtained one and a half inches above the edge of the liver. The blood was examined and showed marked leucocytosis, the red corpuscles did not form rouleaux, and there was well-marked poikilocytosis.

The necropsy was performed twenty-eight hours after death, the body having been in the cold chamber since death. Perforative appendicitis was found, with localised suppurative peritonitis, and suppurative thrombosis of the branches of the superior mesenteric vein, which had spread along other branches and given rise to retroperitoneal abscess. When the abdomen was opened there was an abundant escape of gas. Bubbles of air or gas were noticed in the veins of several of the abdominal viscera. The liver and kidneys floated in water. "The liver was not much above the normal size, and presented rounded edges. Its surface was smooth, with a dark slaty background, mottled over thickly with pale yellowish circular patches 2 to 5 m.m. across. On section, it was extremely emphysematous, and crackled on pressure, and when a lighted match was brought close to the cut surface, and pressure was exerted on the organ, the expelled gas caused a series of explosions. The cut surface had a reddish-brown colour on the

whole, but varied from light red to even a greenish tinge. It exhibited numerous small whitish areas of necrosis, with softened centres, and also numerous sharply-defined bullæ (where the gas had been). These averaged 3 to 5 m.m. across. It was noted that although the mesenteric veins exhibited so extreme a condition of suppurative thrombosis, no thrombi were to be seen in the large vessels of the liver."

The spleen was soft and had an emphysematous feel, but did not present any marked bullæ. The kidneys did not show any marked bullæ, but many whitish areas of necrosis; no abscess formations were found. Large bacilli corresponding in every respect with those described by Welch as the *bacillus aerogenes capsulatus* were found. Sections of the liver, spleen and kidneys showed large colonies of these bacilli. A minute diplococcus was also found on the surface of the media.

Adami looks upon this case as one in which the *bacillus aerogenes capsulatus* commenced to grow before the death of the patient, the development of tympanites the day before death and the localised necrosis in the liver and kidney pointing to an ante-mortem invasion.

CASE 15.—(Jamieson's case.) A. L., a French Canadian, æt. 23, was admitted on April 22nd, 1896, into the Montreal General Hospital. Ten days prior to admission he fell over a wash tub and injured his side and he lost consciousness. Vomiting came on later and lasted for several days. Two days before admission a swelling was noticed in the left side, and he had a severe chill.

Condition on admission. Well nourished. Pulse 84, respiration 24, temperature 98·8°. On the left side a bulging was noticed in the lower thoracic and upper abdominal region; it extended across to the right nipple line and below to the level of the umbilicus. A firm smooth mass was felt in this region, which was dull on percussion. On April 23rd it was aspirated and a few drops of chocolate-coloured fluid were withdrawn, which was found to consist chiefly of blood corpuscles. A diagnosis of hæmatoma with pre-existing kidney disease, was made. The urine contained blood until May 3rd, when it was found to be clear and to contain a trace of albumen. On May 6th he was much worse and was operated on, an incision being made in the left lumbar region. A large amount of gas and dark red putrid fluid was evacuated. The patient died before the end of the operation.

The necropsy was performed twenty-four hours after death. The body was well nourished. The appendix had been removed. "The left kidney was converted into a large sac filled with gas and grumous, foetid and bloody contents." The sac was over twelve inches in its vertical diameter. The kidney was in a condition of putrid pyonephrosis and perinephritis. The right kidney showed evidence of acute parenchymatous nephritis. The spleen was enlarged and crepitant. The liver presented on its surface "numerous greyish brown circular areas the size of pin points, these are sharply defined and the centres in many appear to contain a minute gas bubble. These rapidly increased in number and size when exposed to the air. On squeezing, a brownish frothy blood abundantly exudes. (Liver floated in preserving fluid, and after a few hours was much distended, with abundant froth about it.)" The gall-bladder contained about a drachm of frothy bile. The heart contained a little froth mixed with blood clots.

A microscopical examination of the liver blood showed the presence of bacilli, with mostly square ends, but some with rounded ends about 3 to 5 μ in length. They occurred mostly in pairs and were surrounded by a clear space. A guinea-pig was inoculated intraperitoneally with 1 c.c. of liver blood, and died in about thirty-six hours, with subsequent rapid gas formation. At the end of twenty-four hours the body was blown up to twice its normal size and the tissues and organs were found to be in an emphysematous condition. The microscopical examination showed bacilli similar in form and size to those found in the liver blood. Anaerobic cultures were taken from the human body on two per cent. glucose agar, and after thirty-six hours a growth with gas-formation was obtained. Bacilli in pure culture were found on microscopical examination, corresponding to the description of the *bacillus aerogenes capsulatus*.

Williams points out that there was good evidence of an ante-mortem invasion, as was evidenced by the previously dull tumour becoming tympanitic, and the escape of gas which took place at the time of the operation.

CASE 16.—(Hamilton and Yates.) T. S., æt. 22, a student in arts, was admitted on November 7th, 1896, to the Royal Victoria Hospital for spitting of small quantities of blood, and for spots on the body. The illness started on November 2nd, a few bright red spots of the size of a pin's head being noticed about the left ankle. On the 7th he lost large quantities of blood from his nose, mouth and throat.

Condition on admission. Temperature 102.4°, pulse 100, respiration 24. Hæmorrhages were found all over the body. The blood was sterile: red blood corpuscles 4,840,000; white blood corpuscles 6,000; hæmoglobin 87 per cent. He was under observation for five days, and gradually got worse, delirium supervening just before death, which occurred on the morning of November 13th, eleven days after the onset.

The necropsy was performed ten hours after death. There were marked signs of purpura. The coronary vessels were injected with air; there also appeared to be interstitial emphysema. The right auricle was especially distended with gas. Frothy clots were found in the large vessels. The vessels of the lungs were full of air mixed with blood. Very little gas escaped when the abdominal cavity was opened. "The liver floated easily in water; it was friable in places, emphysematous and somewhat spongy at parts. The surface was smooth, except where air bullæ were plainly visible." The vessels of the spleen contained air, and it floated just below the surface of the water. The right kidney floated in water. The bladder contained some frothy urine.

Coverslip preparations and cultures made at the time of the necropsy showed the presence of the *bacillus aerogenes capsulatus*. The *staphylococcus pyogenes aureus* was obtained from smear-cultures on agar of the heart, spleen, kidney and liver. A bacillus smaller than the colon bacillus was also obtained from the spleen.

The *bacillus aerogenes capsulatus* was not considered to be the primary infection.

CASE 17.—Nicholls reported two cases. (1) A woman, æt. 55, who had had cholecystotomy and cholecystenterostomy performed. The autopsy was performed six hours after death. The blood of the heart contained bubbles of gas and the heart-muscle had a parboiled appearance. Spleen 165 grms.,

soft, with tense capsule; on section, soft, with multiple minute hæmorrhages. Frothy blood exuded from the vessels. Frothy blood also exuded from the vessels of the kidneys. The liver was large, and on section it was soft and had a parboiled appearance. Necrotic areas about the size of a pinhead were found and also a few larger abscesses the size of small marbles. On pressure the exuded blood was found to contain gas. Cultures from all the viscera gave the *bacillus aerogenes capsulatus*. In this case the infection was probably from the intestine into the gall-bladder and then into the liver and other viscera. The gas production in this case was considered to be post-mortem.

CASE 18.—(2) A woman, æt. 53, suffering from strangulated hernia, died under an anæsthetic from inhalation of the gastric contents. The autopsy was performed twenty-seven hours after death. The body was that of a very stout female. Frothy fluid exuded from the nose and mouth. On opening the body the tissues over the sternum crackled and both pus and frothy fluid escaped; gas also escaped from the peritoneal cavity. On the surface of the lungs bubbles of gas could be seen resembling interstitial emphysema. Numerous gas bubbles were seen on the pericardium. The blood in the heart and large vessels contained gas. The spleen weighed 250 grms.; crepitated to the touch; on section, it was pale, soft, and riddled with cavities with ragged walls. The left kidney crepitated to the touch. Liver 3,000 grms.; no crepitation; on section, fatty, pale, and friable; no gas cavities. Microscopical section of the spleen showed numerous cavities and the presence of a large number of large thick bacilli. Section of the kidney showed numerous cavities round which bacilli were massed in large numbers. Anaerobic cultures from the spleen yielded a growth of the *bacillus aerogenes capsulatus*. Aerobic cultures gave a few staphylococci and a small motile bacillus positive to Gram, liquefying gelatine but not gas producing. In this case the *bacillus aerogenes capsulatus* may have gained access during life, but development of gas was certainly after death.

III.—CASES IN WHICH THE *BACILLUS MALIGNI* *ŒDEMATIS* WAS FOUND.

CASE 19.—Bremer, 1888, described a case as one of malignant œdema. A woman, after abortion, developed emphysematous swelling of her right pectoral region and adjacent areas. The autopsy was performed seventeen hours after death. Extensive gas-development was found in the blood-vessels, tissues, thoracic and abdominal viscera, with an enormous number of bacilli devoid of spores in the uterus, blood, organs and serous cavities. The bacilli resembled the *bacillus œdematis maligni*. Guinea-pigs inoculated with sero-sanguineous fluid died in twenty-four hours with lesions similar to those produced by the *bacillus œdematis maligni*.

CASE 20.—Fränkel, in his paper, mentions a case of Wicklein's, in which a micro-organism resembling the *bacillus œdematis maligni* was found.

IV.—CASES IN WHICH THE *BACILLUS* WAS NOT IDENTIFIED.

CASE 21.—Fränkel also mentions a case of Rosenbach's in which a spore-forming bacillus was found, but which could not be cultivated.

CASE 22.—(Bond's case.) Man, æt. 73, who died October 2nd, 1896. Autopsy twenty-two hours after death. No bacteriological examination was made. Beneath the capsule of the liver minute cyst-like projections could be seen. On being handled, this viscus imparted a spongy crepitant sensation,

resembling that of lung, and it floated in water. On section it was seen to be riddled with innumerable cavities, varying in size from microscopic ones to others 6 m.m. in diameter. The walls of the cavities had no definite capsules, and were ragged in appearance.

The pericardium over the left ventricle presented an emphysematous appearance, and was crepitant to the touch. There was no frothing of the blood in the heart. There was no gas formation in any of the organs.

Microscopically there was marked fatty degeneration of the cells and the periphery of the lobules. The liver cells and their nuclei stained badly. The air-containing cavities were irregularly distributed, were devoid of any lining membrane, and appeared to have been formed by a mere bursting asunder of the liver parenchyma. No necrosis or small cell infiltration could be seen. Sections stained with methylene blue showed the presence of large numbers of bacilli among the compressed liver-cells bounding the cavities. No bacilli could be seen in any of the larger bile-ducts, branches of the portal vein, or hepatic artery; a few could be seen in the connective tissue of the portal spaces. Considerable areas of liver tissue were found quite free from bacilli.

There was no clinical evidence suggesting the time of invasion of the micro-organism, and no pathological evidence of the point of invasion.

CASE 23.—(Hebb's case.) A woman, *æt.* 24, was admitted into the Westminster Hospital under Dr. Sturges on April 20th, 1883, for persistent vomiting. She was eight months pregnant, was comatose, and her urine was loaded with albumen. Premature labour was induced by Dr. Potter, and she died on April 27th. The autopsy was performed thirty-six hours after death. The body, with the exception of the lower extremities, was much swollen, partly from *œdema* and partly from decomposition. The liver weighed fifty-two ounces, and in appearance resembled a sponge; it was soft in consistence, of a dirty-yellow colour, and had a honeycombed appearance from the presence of numerous cystoid interspaces which ramified throughout the hepatic tissue. The kidney was similarly affected, but to a lesser degree.

Microscopical examination showed the blood vessels of the liver to be choked with bacilli, which stained not only with any aniline dye, but also with stain which was supposed to be characteristic of the *bacillus tuberculosis*.

The case was brought forward to show that this condition of liver was produced by micro-organisms introduced into the body before death.

The perusal of the preceding twenty-three cases leads to the conclusion that more than one organism is responsible for the production of gas in the internal organs of the body. Of sixteen of these cases in which the *bacillus aerogenes capsulatus* was found in four it was definitely stated that no *bacillus coli communis* was present. In two the *bacillus aerogenes capsulatus* was obtained in pure cultivation, and in two the *bacillus coli communis* was present in conjunction with this organism. In Heydenreich's case the *bacillus aerogenes capsulatus* appeared

to be absent, whereas the organism present was probably the *bacillus coli communis*. In our case we are satisfied that the *bacillus aerogenes capsulatus* was not present, and we feel convinced that the *bacillus coli communis* which we found in pure cultivation was the cause of the production of gas. In one case the *bacillus* of malignant oedema appears to have been the cause, and in four cases the organism is either unknown or undetermined.

The distribution of the gas in the body is extremely variable. In two it is described as being universal. The liver was definitely stated to have been affected in fifteen cases, the spleen and kidneys each nine times, the myocardium six times, the intestine and stomach three times, the penis and scrotum, lungs, cavities of the heart each twice, and once the uterus, suprarenals, urinary bladder, skin of the neck, chest and axilla, serous cavities and coronary arteries. It will be seen that the organ most commonly affected is the liver, and although it is not mentioned in all the cases, in one case (18) it is distinctly stated that the liver was not affected.

Accepting that the gas is produced by one of the organisms previously mentioned, is it possible to arrive at any explanation for this production? At first sight the great preponderance of cases which have affected the liver might bear out the suggestion of Heydenreich that the glycogen in the liver was the most important factor; this view, however, is untenable since Case 18 showed no gas in the liver, although the spleen, kidneys, veins and lungs were all emphysematous.

In the majority of cases two kinds of organisms at least have been found. These have included the *bacillus aerogenes capsulatus* associated with the *bacillus coli communis*, staphylococci, streptococci, or other organisms not designated. It will be seen that the *bacillus aerogenes capsulatus* and the *bacillus coli communis* are both gas producers in artificial media, whereas the other organisms do not produce gas. When either the *bacillus aerogenes capsulatus* or *bacillus coli communis* is associated with the other organisms mentioned, it is quite obvious that it is the cause of the gas production. When

the bacillus aerogenes capsulatus and the bacillus coli communis are found together, it seems to us to be quite probable that each will take its share in the formation of the gas.

Adami says, speaking of the bacillus aerogenes capsulatus : " At the same time it is clear that in this, as, if I mistake not, in all cases, the presence of this gas-producing germ was of the nature of a secondary infection, and indeed I am inclined to doubt whether under ordinary conditions the bacillus can grow in the human organism without the simultaneous presence of aerobic microbes."

This cannot, we think, apply to all cases, since, as we have pointed out in two of the cases the bacillus aerogenes capsulatus was the only organism found in the tissues. We conclude therefore that the bacillus aerogenes capsulatus can grow and produce gas in the tissues of the body without being associated with any other organism. This means that under certain circumstances some of the tissues of the body are under anaerobic conditions. This is not to be wondered at, since, during the præ-agonistic stage, great changes are taking place in the tissues, and it is easy to conceive that the organs and tissues may become virtually free from oxygen on account of the greatly impaired blood supply. While agreeing with Adami as to the doubt that the bacillus aerogenes capsulatus can grow in the human organism under ordinary conditions without the simultaneous presence of aerobic microbes, we maintain that the conditions are not ordinary, and therefore his contention is untenable.

We should think that it is very doubtful whether the bacillus aerogenes capsulatus is really a pathogenic organism in the ordinary acceptation of the term. Pathogenicity being, however, a relative term, it may become pathogenic on account of the lowered vitality and resistance of the patient. A similar phenomenon is found with the proteus vulgaris—this organism is not pathogenic to man, but is sometimes found in thrombi which occur in patients who have suffered for some considerable time from a wasting disease.

On the whole we should consider that the invasion is a præ-agonistic one, and that the development of gas in the tissues during life depends upon the length of the præ-agonistic stage.

We think this opinion is borne out when the causes of death in the twenty-three cases are seen :—

- Case 1. Fatty degeneration.
2. Enteric—third week.
3. Aneurysm of aorta.
4. Recent abortion.
5. Miscarriage of macerated four months' foetus.
6. Peritonitis resulting from traumatic perforation of intestine in hernial sac.
7. Pyelonephritis.
8. Enlarged prostate, catheterisation.
9. Pyæmia.
10. Pyonephrosis.
11. Epilepsy.
12. Enteric—third week.
13. Enteric—second week.
14. Perforative appendicitis with localised suppurative peritonitis.
15. Putrid pyonephrosis, acute parenchymatous nephritis.
16. Purpura.
17. Cholecystotomy and cholecystenterostomy.
18. Strangulated hernia.
19. Abortion.
- 20, 21 and 22. Not stated.
23. Abortion.

It appears to be somewhat strange that in two of the cases at least the *bacillus aerogenes capsulatus* was found to have invaded the tissues whilst the *bacillus coli communis* was absent. Nearly all observers agree that the organism which most frequently and most early invades the moribund tissues is the *bacillus coli communis*. In these two cases, however, strange as it may be, the *bacillus coli communis* had not gained access to the tissues, although the *bacillus aerogenes capsulatus* had done so. This cannot be accounted for from the fact that they will not grow in the presence of one another, since, in artificial media, they do so quite readily, at least for one or two generations.

From the analysis of these cases there appears to be a certain relation between the initial disease and the organs affected, *e.g.*, in case 3 the disease was aneurysm of the aorta and the myocardium was affected; cases 4, 5, and 19 were abortions, and

in all three the uterus and veins were affected ; and in two of the cases the distribution was stated to be universal. This is significant on account of the recognised relation between abortion and septicæmia. On the other hand, although Case 18 would be distinctly described as of intestinal origin, it is equally emphatically stated that the liver was not affected.

BIBLIOGRAPHY.

1. Adami, T. G., 'Upon a Case of Foaming Liver,' Montreal Med. Journal, Aug., 1896, p. 115.
2. Bond, Path. Soc. Transactions, 1898, p. 121.
3. Ernst, P., 'Ueber Einen Gasbildenden Anæroben im Menschlichen Körper und Seine Beziehung zur Schaumleber,' Arch. f. Path. Anat., etc., Berl., 1893, cxxxiii., 308, 338.
4. Fränkel, E., 'Ueber die Ätiologie der Gasphlegmonen (Phlegmone Emphysematose),' Centrallbl. für Bakteriöl. u. Parasitenk 1893, xiii., 13-16.
5. Goebel, C., 'Ueber den Bacillus der Schaumorgane,' Centrallbl. f. Allg. Path. u. Path. Anat., 1895, vi., 465-469.
6. Graham, T. C., Steward, S. H., and Baldwin, T. F., 'The Bacillus Aerogenes Capsulatus,' Case, Diagnosis, Autopsy, and Bact. Study, Columbus M. J., 1893-4, xii., 55-61.
7. Hamilton and Yates, 'An Obscure Case of Purpura Hæmorrhagica with Infection by Bacillus Aerogenes Capsulatus,' Montreal Medical Journal, Aug., 1897, p. 117.
8. Hebb, R. G., Path. Soc. Transactions, vol. xxxv., p. 221.
9. Heidenreich, L., 'Emphysema des Lebers,' Central. für Bacteriologie, xxi., p. 305 et seq.
10. Jamieson, 'Case of Infection by Bacillus Aerogenes Capsulatus,' Montreal Med. Journal, Aug., 1896, p. 119.
11. Nicholls, A. G., 'Notes on Some Cases of Infection by the Bacillus Aerogenes Capsulatus,' British Medical Journal, 1897, ii., 1844.
12. Welch, W. H. and Nuttall, G. H. F., 'A Gas-producing Bacillus (Bacillus Aerogenes Capsulatus, *nov. spec.*) capable of rapid development in the Blood Vessels after Death,' Johns Hopkins Hosp. Bull., 1892, iii., 81-91.

THE COURSE AND SYMPTOMS OF ABDOMINAL ACTINOMYCOSIS.

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ABSTRACT OF THESIS READ NOVEMBER 24TH, 1897, FOR
DEGREE OF M.D. CAMB.

AMONG the many clinical types which Actinomycosis may assume those which occur in connection with the abdominal forms of the disease are perhaps the most obscure, and also, I think, the most interesting.

The following case was under my care from the commencement, and shortly before death was admitted to Mary ward, under Dr. Pye-Smith, to whom I am indebted for permission to publish these notes.

Mrs. K., æt. 55, had enjoyed perfectly good health until December 11th, 1896, when she was attacked with acute abdominal pain. She had no recollection of any illness except a mild attack of influenza three years previously. During the summer of 1896 she paid a visit to America, and returned in apparently good health.

The attack of pain came on during the night, and was very severe; next day the pain persisted, and she looked decidedly ill, but the temperature was normal; the pulse was small and rapid; the bowels were confined, the tongue furred but moist.

The pain was chiefly referred to the epigastrium, and there was some tenderness in the right iliac fossa; there was marked pain on micturition.

Next day the temperature was 99.6° , and this was the highest point reached by the thermometer until ten days before death.

For the next few days the condition remained much about the same; the pulse was quick, out of proportion to the rise of temperature, and the pain was severe.

On December 18th—a week after the onset—the pulse became slower, the tongue cleaner, and there was less pain, but the bowels were still obstinately constipated.

On December 22nd she felt better, and a large enema brought away a considerable quantity of fæcal matter.

For the next few days there were intermittent attacks of severe pain, and the bowels remained constipated.

On December 31st she was up, and felt comfortable.

On January 1st, 1897, was rather purged, and says she passed “blood and slime.” During the night she had a violent attack of pain. The temperature was 98° , pulse 76, and the tongue clean.

January 8th, up and about, but feels weak and has occasional attacks of pain; has passed more “blood and slime.”

During the rest of January she was very weak, but had no attacks of pain; mild aperients were sufficient to keep the bowels open. No blood or mucus was passed during this period.

Early in February anæmia began to be marked, though she declared herself to be better. The spleen was not thought to be enlarged, nor was pain or tenderness over the splenic area noted at any time.

On February 8th there was a sharp attack of pain over the liver, with a short dry cough; the temperature was normal, the liver was not enlarged, nor could any friction sounds be heard.

On this date vomiting commenced, and continued in spite of all treatment for a week. No new symptoms developed until February 21st, when the temperature was raised and there was a rigor. After this date the temperature was very irregular, varying from 98° to 103° .

On February 25th epistaxis occurred, and the anæmia was very marked. The red corpuscles were 75 per cent. of the normal, but the hæmoglobin only 30 per cent., and the white corpuscles were in excess.

The patient's general condition became worse, and petechiæ appeared on the skin; soon a "typhoid" state developed, in which she remained until death took place on March 3rd.

At the post-mortem examination the following state of affairs was found:—The heart and lungs were healthy; the mucous membrane of the small intestine appeared normal and there was no obvious change in the coats; there was a band stretching from a portion of the jejunum to the subjacent ileum. The large intestine was dilated and the walls thinned; in the region of the cæcum it was bound down by some old adhesions. The liver was riddled with small abscesses, and there was one larger one at the portal fissure about three-quarters of an inch in diameter. The portal vein, the superior mesenteric vein, and the splenic vein were thrombosed and filled with foul sanguineous pus. The spleen weighed nineteen ounces; it contained several small, smooth-walled, abscess cavities, evidently of longer standing than those in the liver; these were immediately under the capsule and congregated round the upper branch of the splenic vein. The uterus was small, and there was no endometritis; the right Fallopian tube was bound down to the side of the pelvis by very old adhesions; there was no recent pelvic cellulitis. The pus was not examined under the microscope, but sections through the liver tissue immediately surrounding the abscesses showed the typical appearance of actinomycosis. In spite of numerous examinations of the splenic tissue adjacent to the abscess I was unable to detect actinomyces; the possible importance of this will be alluded to presently. Sections were also made of the splenic vein with a negative result. There was no disease found in any other organ.

Here, then, we have a typical case of abdominal actinomycosis, and one which, from the post-mortem appearances, would be described as originating in the spleen.

The whole illness in the case described above lasted three months, but it will be at once noticed that it really consisted of two distinct illnesses, with an intervening period in which, though perfect health was not restored, there was freedom from the symptoms previously complained of, and the patient herself considered that she was practically well. This is a noticeable feature in most of the cases hitherto described, to which I have had access. Unfortunately, it has not often happened that the same observer has had the care of the case throughout, and frequently it has been only for the second and more severe part of the disease that medical aid has been sought, and, therefore, in published accounts, the earliest symptoms are either very briefly described or altogether omitted. I think this a sufficient reason for discussing in detail symptoms which may appear at first sight to be vague and unimportant, but which become of great interest if they give the least help towards a correct diagnosis. In examining a number of cases there are two symptoms which stand out prominently as characteristic of the earliest stages of abdominal actinomycosis. These are *pain* and *constipation*. Some authors state that diarrhoea may occur instead of constipation, and though it may be readily admitted that it is quite intelligible that the bowel disturbance may occasionally take this form, there is no doubt that constipation, and that of the most obstinate character, is one of the most characteristic symptoms of the early stages of the disease.

With regard to the first of the two characteristic symptoms just mentioned pain is very constant, and also, as a rule, very severe. Thus, I have been able to collect fourteen cases (in addition to my own) in which there is some mention of the early history of the disease, and in twelve of these pain has been a marked symptom; of the other two it is stated that in one the first symptom was "fulness of the stomach," and the patient died after three months without the development of anything more definite; and in the other the patient had been sinking indefinitely for two years.

The pain varies somewhat in character and situation. Thus, in three cases it is described as "colic"; in five others as

indefinite abdominal pain; and in the remaining four the pain resembled that occurring in appendicitis from other causes.

In my own case the pain was a very marked symptom; as far as pain can be gauged it was as severe as that caused by the passage of a renal calculus, but at the same time it could not be definitely localised to any part of the abdomen. In addition to this severe pain there was at times some pain and tenderness in the right iliac fossa, but though at first the case was thought to be one of appendicitis, it was evident that the abdominal pain was quite out of proportion to the other symptoms which might be ascribed to inflammation in that region.

The next symptom to be discussed is the *constipation*. This of course is not a symptom that attracts so much attention as pain, and therefore it is still more difficult to form an opinion as to how often it is present. It is obviously unsafe to draw any conclusions from the small number of cases among those I have collected in which mention is made of the state of the bowels, and therefore we must be content, for the present, to note that in every case where mention is made it is stated that obstinate constipation was present, and further that all observers have agreed that this is a very prominent symptom.

To complete the clinical picture of this stage of the disease, I will just draw attention to one symptom briefly mentioned in the history of the case, namely, the passage of blood and mucus. This is a symptom to which I have found no allusion elsewhere, and—in the absence of hæmorrhoids—it is at least suggestive of some ulceration of the intestine. Quite apart from that, however, it is of value if only to direct a careful attention to the fæces, for if actinomyces be found there, as in a case described by Ransome,¹ a certain diagnosis may at once be made.

Having thus described the condition of a patient in the early stages of abdominal actinomycosis, let us shortly consider the period immediately following them, in which there is restoration of comparative health.

For the same reason that the early indefinite symptoms are often overlooked, or but imperfectly observed, this interval in

¹ B. M. J., 1891, Vol. ii.

which there is almost complete absence of symptoms has hitherto received but scant attention, but it is not without importance in its relation to a true conception of the disease.

In the case detailed above there was distinct improvement after a definite illness lasting a week, and although three weeks elapsed before the patient could be described as convalescent, for the four weeks subsequent to that there was an almost complete absence of symptoms until a definitely new illness commenced with a sharp attack of pain over the liver, followed not long afterwards by severe vomiting and fever.

Very little information can be obtained from published cases of abdominal actinomycosis as to the duration of this interval of health, but one case may be quoted as bearing considerable resemblance to the case described above. Leith² relates the case of a baker, aged 23 years, who was taken ill with severe abdominal pain and subsequent development of a painful swelling in the right iliac fossa. The pain and swelling speedily subsided and in about ten days he resumed work. Shortly afterwards some pain was felt in the right loin and a swelling developed in that region; vomiting followed, and the temperature was raised one or two degrees; constipation was marked throughout. Finally the man died just four months after the first onset of the disease.

This case will be referred to again on account of its importance from the pathological point of view, but at present we are only concerned with the clinical features, and the following points should be noted:—

- (1). The whole illness extended over a period of about four months, but in reality consisted of two illnesses.

- (2). The first attack was short, lasting about ten days, and the chief symptoms were abdominal pain and constipation.

- (3). There was a definite interval during which the patient was able to return to work. The duration of this interval is not stated, but after some little time the patient was again taken ill, and now—

² Edin. Hosp. Rep., 1894.

(4). The second attack was characterised by pain in the lumbar region, vomiting and fever. Death in this case followed an exploratory operation and the immediate cause was acute peritonitis.

I have stated above that the interval between the two attacks was *almost* free from symptoms, but there was one present which must on no account be overlooked, namely, progressive anæmia.

It has generally happened that cases of actinomycosis affecting the abdominal viscera have only come under observation when the more serious symptoms, to be presently described, have already developed, and, therefore, it is not surprising if the presence of anæmia has been overlooked.

In the case under my own observation the anæmia began to be marked some time before the onset of any serious symptoms, and was steadily progressive until it reached such a degree as to cause epistaxis, and hæmorrhages under the skin.

[Since the above was written, two very interesting cases have been described by Makins in the St. Thomas's Hospital Reports, and in one of these the anæmia was a marked feature of the case.]

I have been able to find only one other case in which special attention was paid to the anæmia, or in which examinations of the blood were made. This case, recorded by Latimer and Welch,³ was that of a labourer, aged 21, who was stated to have had three attacks of appendicitis during 1894. When seen in November, 1895, he was suffering from anæmia, headache, loss of appetite, and obstinate constipation. The temperature varied during his illness from 98° in the morning to 103° in the evening; the liver was felt to be enlarged, but nothing else abnormal was detected in the abdominal organs.

The anæmia became more marked, and anasarca and dyspnoea developed. The number of red corpuscles was 64 per cent. of normal. The proportion of red to white blood corpuscles was thirteen to one, and the hæmoglobin was 25 per cent. of the normal. The patient became gradually feebler, and died three

³ Trans. Ass. Am. Phys. and Surg., 1897.

months after the date on which he was first seen. The post-mortem showed the disease to be actinomycosis of the liver.

This case was considered to be one of leuchæmia, and as in the case which forms the text of this paper, it will be noticed that the characteristics of the blood are :—

- (1). A slight diminution in the number of the red corpuscles.
- (2). A considerable diminution in the quantity of hæmoglobin in each corpuscle.
- (3). An excess—in the one case a very large excess—in the number of the white corpuscles.

Whether further investigation shows these features to be constant or not the fact remains that pronounced anæmia of a very definite type may occur during what I may call the latent period of the disease, and, in fact, may be the first warning that the patient, who was thought to be convalescent, is really on the verge of a very serious if not fatal illness.

Without laying too much stress on this one symptom of anæmia it is perhaps not too much to say that should an investigation of the blood reveal such features in a patient who had recently suffered from severe abdominal pain accompanied by constipation the possible occurrence of actinomycosis of one or more of the abdominal viscera must be constantly borne in mind.

So far I have described the first illness and subsequent latent period as if the symptoms and duration were constant in these stages.

This is in the main true, but there are two points in which the disease may depart from the type described above. These are, firstly, that the original attack may exactly simulate ordinary appendicitis, and secondly, that the latent period may be very much prolonged.

The form of the disease characterised by these features is indeed the one more commonly met with, and it would be easy to collect many cases described as recurrent appendicitis in which, after an interval of many months, or even years, the symptoms of abdominal actinomycosis have developed.

These symptoms group themselves naturally under two headings—those pointing to abdominal suppuration, and those associated with tumour formation.

The case I have detailed above illustrates very well the first type; clinically it is an abdominal pyæmia, and presents no special features which call for discussion or which aid diagnosis. Cases such as this are rarely recognised before death, and it is only, I think, by a careful consideration of the early history, with a systematic examination of the stools and blood, that we can hope to arrive at a correct conclusion.

The second type of case presents a far less difficult problem, inasmuch as its characteristic feature is tumour formation. This is the form more commonly assumed by the disease when it has previously caused attacks of what is doubtless actinomycotic appendicitis. Accordingly we find that the tumour which forms is most frequently situated in the right iliac fossa, but it is by no means rarely in the lumbar region that the disease makes itself evident, forming the lumbo-abdominal group of Cornil and Babes, and occasionally the abscess points in the epigastric, umbilical or some other region of the abdomen.

The exact position of the tumour is, however, a matter of secondary importance, since sooner or later nature or the surgeon affords an exit for the pus, and this gives an opportunity at the same time for both diagnosis and treatment to be made complete.

The prognosis in these cases is accordingly much more hopeful, and there are not a few recorded where free incision, followed perhaps by repeated scraping, has led to a complete recovery.

This completes the clinical picture of the disease, and we turn to a study of the morbid anatomy of our original case in the hope of finding an explanation of some of the symptoms we have observed.

The spleen may be taken first as being the most obviously diseased organ found at the post-mortem examination.

As already stated, the abscess cavities were lined with smooth walls of some thickness—characters associated with abscesses of some standing. The exact age of the abscesses cannot be determined, but the important point to grasp is that these were

evidently less recent than those in the liver. At the time of the autopsy the pus was not examined under the microscope, but no unusual characters were noticeable to the naked eye. As I have already stated, I examined a fair number of sections of the splenic substance bordering on the abscess, but was unable to detect the "ray fungus." As this was so easily detected in corresponding situations in the liver, I think we may fairly assume that the fungus which was originally present in the spleen had been destroyed, and that there is therefore in the solid viscera the same natural tendency to cure as will be shown to exist in the intestine.

With regard to the position of the abscess, it is important to notice that it was not deep in the substance of the spleen but close under the capsule.

I have not been able to find many cases recorded where the spleen has been the site of an actinomycotic abscess, nor indeed is the mere fact of any one organ being so affected of any great importance, but Bostrom⁴ gives a good illustration of a spleen affected with actinomycosis in an early stage, and in that case it is quite obvious that the disease is gradually invading the spleen from without. This I think also occurred in the case under discussion, though the exact path of the organism from the bowel to the spleen cannot be demonstrated.

The condition of the liver does not call for any particular comment. Actinomycotic abscesses in the liver have been so often met with and described that the typical honeycombed appearance is quite familiar. In this particular case, however, the liver did not have that typical appearance, but rather resembled the condition known as "multiple or pyæmic abscesses of the liver." A little consideration, however, is quite sufficient to explain this; the large honeycombed abscess occurs in those cases generally described as primary actinomycosis of the liver, while in the case under consideration the condition of the liver was, I think, undoubtedly secondary to that of the spleen, the sequence of events being that the ray fungus traversing the peritoneum infected the spleen where secondary suppuration

⁴ Untersuchungen über die Aktinomykose des Menschen.

took place; this spread to the splenic vein, and so to the portal vein, and thus the liver received at the same time a double infection of pyogenic and actinomycotic organisms.

The intestines to the naked eye appeared normal and no microscopic examination was made. There was in addition to what has been already described slight thickening of the vermiform appendix but it could not be definitely stated that there had been previous appendicitis.

The resemblance between the course of events as here described and those following infection by the ova of *tænia* of *echinococcus*, is very striking, and indeed in many points actinomycosis is the exact analogue of hydatid disease. Actinomycosis has occurred in nearly every part of the alimentary canal, but it is only rarely detected while confined to that part. Thus, the familiar form of the disease occurring in the tissues of the neck is in the majority of cases secondary to a primary infection of the mouth; while œsophageal actinomycosis has occurred sufficiently often to have been made the subject of a thesis by Gaede⁵. Again, the stomach may be the organ first affected, as in a case described by Grill,⁶ where an empyema of the left side in a man aged 26, led to the formation of pleuro-cutaneous fistulæ and ultimately death ensued. The autopsy showed the primary disease to be in the wall of the stomach, and the pleura and lung were secondarily affected through the diaphragm. Proceeding down the alimentary canal we find many cases recorded—as by Ransome,⁷ Chiari,⁸ Israel,⁹ Leith¹⁰ and others—while the small and large intestine have been primarily invaded; and finally the anus has been found by Large¹¹ to have yielded to an attack which had presumably been successfully resisted by the whole of the rest of the alimentary canal.

⁵ Thèse de Lyon, 1896.

⁶ *La Semaine Médicale*, Sept. 4, 1896.

⁷ *B. M. J.*, 1891, Vol. ii.

⁸ *Prag. Med. Woch. Schr.*, 1884, No. 10.

⁹ *Path. Soc. Trans.*, Vol. 36.

¹⁰ *Loc. cit.*

¹¹ *Annals of Surg.*, Sept., 1896.

This case is of special interest, inasmuch as the chronic nature of the disease and the part of the body affected enabled complete extirpation to be carried out, and a cure effected.

There was a fibrous mass surrounding the lower part of the bowel and containing multiple foci of actinomycosis; one was quite close to the bowel, but seemed entirely shut off from it by a hard cicatricial wall.

It will thus be seen that any part of the alimentary canal from mouth to anus, may be successfully attacked by the ray fungus, the probable explanation being that the micro-organism is only able to effect an entrance after an abrasion of the mucous membrane has taken place. This is actually found to be the case with animals, since feeding them with the fungus is not sufficient to produce the disease (Grill, *loc. cit.*). Let us now consider the pathology of the disease as it appears in the alimentary canal. Israel describes the two forms of primary actinomycosis of the intestine :—

- (1). Non-destructive superficial disease of the mucous membrane.
- (2). Destructive parenchymatous progressive actinomycosis.

With reference to the first form it may be fairly supposed that in some cases a natural cure may be effected. Such an event, for instance, may have occurred in the case described by Ransome (*loc. cit.*) where the fungus was found for a time in the stools, but afterwards completely disappeared and the patient went home apparently well.

With reference to the destructive form, in the well-known case described by Chiari (*loc. cit.*), there was no active ulceration going on at the time of death, but it was found that the fungus filaments had grown into the intestinal glands.

Speaking of this case Roswell Park¹² says, "In such cases generally small nodules about the size of a pea may be found in the submucous tissue and the mucosa; they soften and form ulcers with determined edges, their bases reaching the deepest layer. These may undergo cicatrisation, but generally ulceration extends through the peritoneum." There is, however, another

¹² *Annals of Surg.*, 1891.

sequel, perhaps the most important of all, namely, a restoration of the mucous membrane to its normal condition, while beneath it foci of actinomycosis remain in an active condition. This was found in a remarkable case described by Leith and already referred to; Leith found that the tissue between the small submucous abscesses showed a tendency towards cicatrisation, and came to the conclusion that there was a natural tendency towards extrusion of the disease into the bowel.

So far then it is clear that actinomycosis may attack any part of the alimentary canal with one of the four following results :—

(1). Complete recovery of mucous membrane without affection of deeper layers.

(2). Recovery of mucous membrane leaving active disease in the submucous tissue.

(3). Ulceration of the mucous membrane with subsequent cicatrisation.

(4). Perforation of the bowel.

One of the most striking features of the post-mortem accounts of cases of the disease is the frequency with which the peritoneum has been described as thickened or some portion of the intestines as matted together. Unfortunately, in many cases, especially where suppuration has taken place, the thickening is so extensive that little information can be gained as to its origin and course, and it is to the slighter cases we must turn for help. Here, again, we are met by the difficulty that, as hitherto no particular attention has been paid to this point, the condition of the peritoneum in the cases which would be of greatest value has received but scant attention. I will, therefore, briefly describe a few cases which appear to me to throw some light on this point.

In Ransome's case during life a mass could be felt in front of the rectum close to the prostate; laparotomy was performed, and the mass was found to be a coil of small intestine bound down by adhesions. Subsequently the fungus which had previously been present in urine and fæces disappeared from the latter, though continuing to be present in the former, and at the same time the patient was relieved of the symptoms from which he had suffered. Here there seems no doubt that actinomyces had primarily

affected the mucous membrane of the small intestine and had then passed through the wall and gradually invaded the prostate.

Another suggestive case is recorded by Israel (*loc. cit.*). Here scattered pigmented and cicatrised spots were found on the inner surface of the small intestine; there was an actinomycotic tumour in the left ovary, and the abdominal and pelvic organs were universally adherent.

Bostrom (*loc. cit.*) describes and illustrates a case where actinomycosis of the intestine had spread to one ovary leaving a fistulous connection between the two viscera. In Leith's case there were firm fibrous adhesions round the cæcum and ascending colon, and the liver was the seat of typical actinomycotic abscesses.

Lange (*loc. cit.*) reports a case where dense infiltration of the iliac fossa led to an incision being made, with the result that the disease was found to be actinomycosis. Subsequently a fistula formed in the ascending colon, which was surrounded by dense fibrous tissue. The portion of bowel was dissected out and resected with the result that complete recovery ensued.

Finally, I may refer to the case described at length by Latimer and Welch, and to which reference has already been made. Here the peritoneum was found generally thickened, and the intestines bound together by adhesions. The entire ascending colon and hepatic half of the transverse colon were massed in "inflammatory" tissue. The right lobe of the liver was occupied by a mass which extended through the whole thickness of that organ, but occupied a larger transverse area in the lower two-thirds than in the upper third. The inferior surface and posterior margin of the right lobe were connected with a dense mass of fibrous adhesions which included the hepatic vessels, right adrenal gland, and hepatic flexure of the colon. Finally, the authors concluded that the distribution of lesions and their appearance made it probable that the process started in the appendix vermiformis, crept up the tissues, round the ascending colon, and invaded the liver at the lower surface.

I am aware that a few cases such as these cannot be taken to prove very much, but in the absence of any more definite knowledge on the point they are at least very suggestive of the following points :—

(1). In the absence of suppuration there is a definite formative process going on whereby the peritoneum becomes thickened and fibrous tissue produced.

This process is similar to that which occurs in the neck and other parts of the body which may be the seat of actinomycosis.

(2). There is a relation between the part of the bowel affected and the organ which becomes secondarily diseased.

Thus we have seen that the ovary or prostate may be the seat of an actinomycotic abscess when the small intestine has been the original focus. Such cases are, however, infrequent compared with those in which the liver has been found after death to be affected, and equally so with those in which the symptoms and signs during life have pointed to the appendix. The connection between appendicitis and subsequent disease of the liver is an anatomical one, namely, the presence of an abundant supply of subperitoneal tissue around the ascending colon which thus affords a well-provisioned road for the progress of the fungus.

(3). The actinomyces may remain latent, yet active for a very long time in the thick fibrous tissue surrounding the bowel.

Grainger Stewart has said that the disease has been met with in the wall of the intestine, in the peritoneum, in the retroperitoneal tissue, and often in the substance of the liver, and this unconsciously I think very aptly expresses its most common path of progress.

But we must not forget that, as Israel says, "It is not a little remarkable that no trace of the migration of an actinomycotic tumour can usually be detected between the point of its indubitable origin and the point where it comes to light;" we must therefore, not expect to be able in every case to trace with certainty the path of the disease either by symptoms during life or by post-mortem appearances.

Still, in spite of difficulties caused partly by the nature of the disease, and partly by the inaccuracy of our observations of

clinical and pathological phenomena, we have, I think, a sufficient number of facts ready to hand to enable us to understand the life history of the ray fungus within the human body.

I will endeavour to avoid repetition as far as possible in the following brief description of the course of events:—The fungus enters by the mouth and passing into the intestine finds a resting place wherever any slight abrasion may have occurred. Should circumstances be unfavourable to the growth of the fungus a slight catarrhal inflammation results, the micro-organism is cast off, and the patient recovers. As a rule, however, the fungus passes more deeply into the submucous tissue and the inflammatory reaction is more severe. There is still, however, a possibility that the formation of a small abscess may lead to the extrusion of the fungus leaving the bowel intact, but for the formation of a small cicatrix; more often, however, by this time the filaments of the actinomyces have grown into the deeper coats of the intestine, and slowly, but surely, proceed on their course of destruction.

A long period may now elapse during which the patient is enjoying comparative health; meanwhile the fungus is gradually growing in the direction which is found to be most favourable, and frequently this is determined by the presence of some tissue such as the sub-peritoneal tissue with its varying distribution. At present there is no pus-formation, no lymphatic infection or vascular metastases in distant organs, but after a period, which may be weeks, or may be years, the disease again lights up afresh. It has now invaded a solid viscus, and suppuration has taken place; the course of the disease is now rapid, pyogenic organisms become associated with actinomycotic, a general pyæmia is produced and death rapidly follows.

There is, however, an alternative course for the disease to follow, and this must now be considered. Should the intestinal infection be severe, ulceration proceeds through the various coats and a fistula is formed; in these cases the ray fungus is from the first associated with pyogenic organisms, and there is an early formation of abdominal abscesses; these are as a rule shut off from the general peritoneal cavity, and follow the usual course of

such abscesses; surgical aid is generally sought early, and is frequently able to save the patient's life.

Such, then, is the general course of abdominal actinomycosis, but as nearly every rule has its exceptions, so here on rare occasions the abdominal cavity may be infected through the vagina, or even through the skin. The possibility of such an event occurring is shown by cases recorded by Giordano of Venice¹³ and by Delepine¹⁴ even if we except the cases of Grainger Stewart¹⁵ and Zemann.¹⁶ I do not, however, propose to make any further allusion to these cases, since they do not affect the point at issue.

In conclusion, I may sum up the chief points which I have endeavoured to prove :—

(1). Actinomycosis of the abdomen as ordinarily observed is what may be called a residual disease—comparable to the residual abscesses described by Sir James Paget as “suppuration among the products of a former inflammation,” and bearing a striking analogy to the hydatid stage of echinococcus disease.

(2). In the great majority of cases the seat of primary infection is in the intestine, and rather more frequently in the appendix vermiformis.

(3). There is clinical as well as experimental evidence that the disease is essentially formative and not destructive, and that pus formation does not occur in the absence of other organisms.

(4). The disease spreads from the intestine to one or more of the abdominal viscera by direct continuity of tissue.

(5). The original disease has a definite entity of its own, the chief symptoms being abdominal pain, constipation and anæmia.

¹³ *Annals of Surg.*, 1896.

¹⁴ *Trans. Path. Soc.*, 1889.

¹⁵ *Edin. Hosp. Rep.*, 1893.

¹⁶ *Wien. Med. Jahrb.*, 1883.

LIST

OF

GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,

&c., &c.,

IN THE YEAR 1897.

University of Oxford.

Final Examination for the Degrees of Bachelor of Medicine and Surgery.

R. Howard.

University of Cambridge.

Final Examination for the Medical and Surgical Degrees.

Part I.

E. R. Delbruck.	F. J. Lidderdale.	R. Mathias.
C. Jephcott.	W. J. Lindsay.	P. R. Lowe.

Part II.

E. R. Delbruck.	W. J. Lindsay.	A. E. Porter.
F. A. Godson.	R. Mathias.	C. C. Stead.
C. Jephcott.	C. E. Michael.	C. A. Trouncer.

Second Examination for the Medical and Surgical Degrees.

Part I.

H. Davies-Colley.

Part II.

J. M. Brydone.	C. H. Glenn.	N. F. Ticehurst.
H. Davies-Colley.	T. P. Thomas.	D. P. Watson.

First Examination for the Medical and Surgical Degrees.

Part I.

H. A. Cutler. | A. G. Harvey.

Examination in Sanitary Science.

G. T. Cattell.	G. G. Genge.	B. B. Ham.
	A. T. McConkey.	

University of London.

Examination for the Degree of Doctor of Medicine.

A. Chaning-Pearce.		C. J. Harnett.
		T. M. Thomas.

Examination for the Degree of Master in Surgery.

F. J. Steward.		E. C. Taylor.
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Examination for the Degree of Bachelor of Surgery.

First Division.

C. H. Fagge.

Obtained the Gold Medal and Moiety of Scholarship and First-class Honours.

Second Division.

E. Fisk.		G. K. Levick.		W. T. Milton.
C. R. Hodgson.		G. E. Manning.		G. E. Richmond.
		P. N. Vellacott.		

Examination for the Degree of Bachelor of Medicine.

First Division.

R. H. Ashwin.

Second Division.

E. S. Dukes.		E. Fisk.		S. Whicher.
A. Earnshaw.		J. Moore.		

October.

Second Division.

C. H. Fagge.

Obtained the Gold Medal and Scholarship and First-class Honours in Medicine, and Honours in Forensic Medicine.

R. W. Mayston.

Obtained Honours in Forensic Medicine.

W. T. Milton.

Obtained First-class Honours in Obstetric Medicine and in Forensic Medicine.

G. E. Richmond.

Obtained Honours in Forensic Medicine.

H. J. Starling.

Obtained First-class Honours in Forensic Medicine.

H. W. Bruce.		C. R. Hodgson.		G. K. Levick.
E. Coleman.		H. Leader.		J. H. Phipps.
		J. T. Roberts.		

Honours in Medicine.

W. P. Walker.

Intermediate Examination in Medicine.

January.

Entire Examination.

Second Division.

G. N. Meachen.

H. M. Reeve.

Excluding Physiology

Second Division.

A. Fraser.

W. W. Harrison.

A. Moon.

B. W. Moss.

W. G. Stewart.

Physiology only.

First Division.

W. B. Secretan.

Second Division.

A. H. Carter.

H. E. C. Fox.

G. C. Owsley.

July.

Honours Examination.

J. F. Northcott.

Obtained the Gold Medal and First-class Honours in Physiology and Histology.

E. A. Miller.

Obtained Honours in Physiology and Histology and in Materia Medica and Pharmaceutical Chemistry.

J. Atkins.

Obtained First-class Honours in Materia Medica and Pharmaceutical Chemistry.

Entire Examination.

Second Division.

J. A. Butler.

P. W. L. Camps.

D. G. Greenfield.

G. Lewin.

G. T. Wrench.

H. McD. Parrott.

E. Stott.

Excluding Physiology.

First Division.

A. W. Penrose.

Second Division.

P. T. Manson.

O. Marriott.

Physiology only.

Second Division.

A. Densham.

E. C. B. Ibotson.

B. W. Moss.

E. E. Parrett.

W. G. Stewart.

Preliminary Scientific (M.B.) Examination.

January.

Entire Examination.

First Division.

C. H. Dawe.		W. M. Robson.
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Second Division.

C. E. Adams.

Chemistry and Experimental Physics.

A. J. Beadel.		L. J. Hughes.		F. C. Robinson.
G. B. F. Churchill.		E. T. Kendon.		M. D. Wood.
W. H. Cole.		G. S. Robertson.		

Biology.

G. T. Collins.		J. Evans.		H. C. Keates.
M. A. Collins.		A. C. H. Gray.		W. G. Parker.
		F. L. Thomas.		

July.

Entire Examination.

Second Division.

H. M. Goldstein.		C. H. Robertson.		A. H. E. Wall.
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Chemistry and Experimental Physics.

K. Anderson.		P. W. Hamond.		L. G. Nash.
J. Braithwaite.		A. W. Iredell.		

Biology.

W. H. Cole.		W. C. Lewis.		F. C. Robinson.
L. J. Hughes.		G. S. Robertson.		

Intermediate Examination in Science and Preliminary Examination conjointly.

First Division.

W. E. J. Tuohy.

Honours Candidate recommended for a Pass.

A. E. H. Pakes.

B.Sc. Examination.

First Division.

E. A. Miller.

Second Division.

L. E. Stamm.

LL.B. Examination.

Second Division.

H. Nolan.

University of Durham.

Examination for the Degree of Doctor of Medicine.

W. Harland Peake. | O. Beven.

Examination for the Degree of Doctor of Medicine for Practitioners of Fifteen Years' standing.

T. Reuell Atkinson.

Second Examination for the Degree of Bachelor of Medicine.

G. Burton-Brown.

Obtained Honours.

H. Braund. | R. T. Brown. | A. M. Thomas.
T. J. A. Tulk-Hart.

First Examination for the Degree of Bachelor of Medicine. Chemistry and Physics.

R. T. Brown. | G. Burton-Brown. | F. W. Sime.
T. J. A. Tulk-Hart.

Elementary Biology.

O. C. H. L. Moll.

Royal College of Physicians.

Admitted to the Membership.

A. P. Beddard. | M. Craig. | F. H. Edwards.

Final Examination for the License.

January.

C. H. Booker.	J. Moore.	H. J. Thomas.
H. Fulton.	L. G. Reynolds.	T. J. A. Tulk-Hart.
F. A. Godson.	E. T. Scowby.	J. P. Watkins.
H. R. Marsh.	P. G. Temple.	S. Whicher.
R. W. Mayston.	E. R. Thomas.	

April.

A. B. Carter.	N. Y. Lower.	R. B. Stamford.
E. F. Clowes.	C. E. Michael.	M. E. Tresidder.
H. E. Cock.	A. E. Porter.	C. P. Weekes.
C. R. Evans.	P. C. Prince.	J. C. Williams.
H. Fulham-Turner.	A. M. Rygate.	

July.

A. R. Adams.	H. W. Dudgeon.	R. Kay.
H. W. Allan.	G. E. Duncan.	W. Mussellwhite.
F. S. Batchelor.	W. N. East.	H. V. Smith.
H. Cardin.	F. C. Hitchins.	L. H. Y. Stephen.
C. L. G. Chapman.	C. R. Hodgson.	P. Turner.
A. J. Cleveland.	S. Hughes.	M. H. Way.
F. H. L. Cloud.	A. J. Hull.	

October.

J. D. W. Beavis.	B. B. Gough.	H. J. Starling.
A. T. Falwasser.	A. E. Hancock.	M. H. Thornely.
R. T. Fitz-Hugh.	C. H. Mossop.	J. G. Watt.
F. E. Fremantle.	C. C. Poole.	

Royal College of Surgeons.

Final Examination for the Fellowship.

H. A. Duffett.	W. S. Handley.	T. M. Thomas.
C. H. Fagge.	G. E. Manning.	

Primary Examination for the Fellowship.

D. S. Graves.	J. F. Northcott.	G. S. Simpson.
C. T. Hilton.	W. B. Secretan.	

Final Examination for the Membership.

January.

C. H. Booker.	J. Moore.	H. J. Thomas.
H. Fulton.	L. G. Reynolds.	T. J. A. Tulk-Hart.
F. A. Godson.	E. T. Scowby.	J. P. Watkins.
H. R. Marsh.	P. G. Temple.	S. Whicher.
R. W. Mayston.	E. R. Thomas.	

April.

A. B. Carter.	N. Y. Lower.	R. B. Stamford.
E. F. Clowes.	C. E. Michael.	M. E. Tresidder.
H. E. Cock.	A. E. Porter.	C. P. Weekes.
C. R. Evans.	P. C. Prince.	J. C. Williams.
H. Fulham-Turner.	A. M. Rygate.	

July.

A. R. Adams.	H. W. Dudgeon.	R. Kay.
H. W. Allan.	G. E. Duncan.	W. Mussellwhite.
F. S. Batchelor.	W. N. East.	D. F. Roberts.
H. Cardin.	F. C. Hitchins.	H. V. Smith.
C. L. G. Chapman.	C. R. Hodgson.	L. H. Y. Stephen.
A. J. Cleveland.	S. Hughes.	P. Turner.
F. H. L. Cloud.	A. J. Hull.	M. H. Way.

October.

J. D. W. Beavis.	B. B. Gough.	H. J. Starling.
A. T. Falwasser.	A. E. Hancock.	M. H. Thornely.
R. T. Fitz-Hugh.	C. H. Mossop.	J. G. Watt.
F. E. Fremantle.	C. C. Poole.	

Final Examination for the License in Dental Surgery.

May.

C. E. Brown.	S. Harrison.	E. W. Smith.
E. C. Brown.	R. G. S. Holmes.	A. St. J. Styer.
P. H. Cook.	J. M. C. Jacobs.	E. G. W. Wallis.
A. Cooper.	A. E. Oddy.	C. A. Wilson.
W. Floyd.	E. Picnot.	

November.

G. O. Betts.	H. L. Darrell.	S. J. Redpath.
T. W. Bromley.	E. B. Dowsett.	V. G. Smith.
H. A. G. Butler.	A. M. Kempe.	C. B. Stainer.
H. A. E. Canning.	A. Kendrew.	B. J. Sumerling.
J. K. Clark.	G. Marshall.	A. W. Walker.
F. E. Corin.	E. H. Musgrove.	F. Ward.
J. M. P. Crombie.	G. A. Pedley.	W. Wilmore.

Society of Apothecaries of London,

Final Examination for the License.

J. T. Brickwell.	H. R. Marsh.	C. C. Poole.
H. Fulton.	E. J. O'Meara.	W. F. Reckett.
A. J. Hull.	J. Ponsonby.	C. A. Trouncer.

MEDALLISTS AND PRIZEMEN.

JULY, 1898.

Open Scholarships in Arts.

Percy Reginald Bolus, Bancroft School, Woodford Wells, £100.
 Gayton Warwick Smith, Liverpool College, Certificate.

Dental Students.

Percival Pasley Cole, Weymouth College, £30.

Open Scholarship in Science.

Albert Ernest Hardman Pakes, Guy's Hospital, £150.

Scholarship for University Students.

Aubrey Hugh Davies, Caius College, Cambridge, £50.
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F. E. Hutchinson.	L. E. C. Handson.	R. Balderston.
C. R. Nicholson.	A. R. Thomas.	A. J. Wernet.
H. N. Clarke.	V. T. C. Bent.	T. W. S. Browne.
H. A. Gaitskell.	J. B. C. Brockwell.	G. N. Meachen.
E. Fryer.	J. H. Jones.	A. G. Osborn.
F. H. R. Heath.	F. D. Turner.	A. C. Ambrose.
C. D. Outred.	W. C. Pritchard.	E. Ashby.
A. T. Falwasser.	H. R. H. Denny.	F. Shuffelebotham.
A. D. Lewis.	S. A. Ruzzak.	A. H. B. Kirkman.
P. W. Moore.	H. C. Holden.	F. W. Brook.

DENTAL SCHOOL.

APPOINTMENTS HELD DURING THE YEAR 1897.

DENTAL HOUSE-SURGEONS.

K. W. Goadby.	G. S. Simpson.	J. A. M. Donston.
E. C. Brown.	W. Floyd.	C. A. Wilson.

ASSISTANT DENTAL HOUSE-SURGEONS.

W. Floyd.	C. A. Wilson.	A. D. Hopkins.
S. J. Redpath.	G. O. Betts.	B. J. Sumerling.
J. K. Clark.	R. Umney.	

DEMONSTRATORS IN THE CONSERVATION ROOM.

G. O. Betts.	T. W. Bromley.	J. K. Clark.
A. C. Carpenter.	R. Umney.	W. Wilmore.
C. H. Huckle.	T. H. Wilkin-on.	P. S. Campkin.
J. S. Barnfield.	E. N. Mason.	T. F. Ryan.

ASSISTANT DEMONSTRATOR OF DENTAL MICROSCOPY.

E. H. Musgrove.

DRESSERS IN THE EXTRACTION ROOM.

C. S. Brookhouse.	C. H. Huckle.	D. P. Tracy.
A. A. Bartholomew.	F. W. S. Stone.	E. P. Uttley.
J. Harper.	J. S. Farnfield.	L. M. Fleetwood.
J. R. S. Ash.	T. F. Ryan.	L. C. A. Knight.
A. L. Lambert.	E. H. Musgrove.	E. N. Mason.
C. F. Jessop.	H. W. Morris.	P. E. Chandler.
H. H. Evans.	J. G. McAlpin.	W. H. Phillips.
A. Hughes.	A. J. Gwatkin.	G. J. Lewis.
S. H. Ölver.	C. F. Witcomb.	P. S. Campkin.
E. G. Walton.	E. C. Bartlett.	J. S. Biss.
S. H. Jones.	W. H. Loosely.	W. W. C. Jones.
P. H. H. Palmer.	A. E. Rowlett.	F. Warlow.
E. B. L. White.		

DRESSERS IN THE GAS ROOM.

C. B. Stainer.	B. J. Sumerling.	S. J. Redpath.
F. Ward.	W. E. Ganney.	A. W. Walker.
A. Cooper.	A. Kendrew.	C. E. Brown.
W. N. Mitchener.	H. L. Dorrell.	E. Stringfellow.
J. Harper.	G. A. Pedley.	F. W. S. Stone.
C. S. Brookhouse.	J. K. Clark.	D. P. Tracy.
W. Wilmore.	R. Umney.	F. E. Corin.
E. P. Uttley.	E. H. Musgrove.	T. H. Wilkinson.
B. B. Westlake.	E. B. Marshall Frost.	A. C. Carpenter.
A. A. Bartholomew.	T. F. Ryan.	R. P. Millett.
E. C. Bartlett.	E. N. Mason.	P. S. Campkin.
C. T. Witcomb.	L. M. Fleetwood.	H. A. G. Butler.
A. L. Lambert.	H. W. Morris.	S. H. Olver.
C. F. Jessop.	J. S. Farnfield.	H. Charnock.
A. J. Gwatkin.	E. G. Walton.	M. P. Nathan.
C. H. Huckle.		

DRESSERS IN THE CONSERVATION ROOM.

A. O. Bell.	H. A. C. Canning.	S. Harrison.
C. H. Huckle.	T. E. Norton.	E. Stringfellow.
F. W. S. Stone.	H. A. G. Butler.	F. Curtis.
J. Harper.	R. P. Millett.	A. L. Rowley.
A. W. Walker.	P. H. Cook.	E. B. M. Frost.
A. M. Kempe.	W. H. Loosely.	E. H. Musgrove.
T. H. Wilkinson.	H. L. Dorrell.	B. F. Henry.
R. G. S. Holmes.	A. Kendrew.	T. F. Ryan.
C. B. Stainer.	G. Marshall.	J. M. P. Crombie.
C. Lee.	G. D. Slater.	V. G. Smith.
F. Ward.	G. J. Lewis.	S. W. Cock.
A. Cooper.	F. E. Corin.	C. E. Brown.
D. P. Tracy.	W. Wilmore.	C. S. Brookhouse.
P. E. Chandler.	A. J. Gwatkin.	M. P. Nathan.
E. P. Uttley.	E. G. Walton.	D. G. Worts.
A. C. Carpenter.	H. H. Evans.	W. E. Ganney.
L. C. A. Knight.	J. G. McAlpin.	W. H. Phillips.
C. F. Witcomb.	A. A. Bartholomew.	J. S. Farnfield.
E. N. Mason.	C. F. Jessop.	S. H. Olver.
E. W. Smith.	H. Knight.	J. R. S. Ash.
P. S. Campkin.	A. Hughes.	A. L. Lambert.
H. W. Morris.	R. Umney.	L. M. Fleetwood.
H. Charnock.	B. J. Sumerling.	A. Battersby.
E. R. Howlett.	F. Warlow.	E. B. L. White.
P. H. H. Palmer.	W. W. C. Jones.	H. W. Morris.
E. C. Bartlett.	A. E. Rowlett.	J. G. Biss.
J. A. Whittington.	W. Jarvis.	S. H. Jones.

GUY'S HOSPITAL.

MEDICAL AND SURGICAL STAFF.

1900.

Consulting Physicians.—SIR SAMUEL WILKS, BART., M.D., LL.D., F.R.S.;
F. W. PAVY, M.D., LL.D., F.R.S.; P. H. PYE-SMITH, M.D., F.R.S.;
J. F. GOODHART, M.D., LL.D.

Consulting Surgeons.—J. BIRKETT, Esq.; THOMAS BRYANT, M.Ch.

Consulting Obstetric Physician.—H. OLDHAM, M.D.

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FREDERICK TAYLOR, M.D.

H. G. HOWSE, M.S.

W. HALE WHITE, M.D.

R. CLEMENT LUCAS, B.S.

G. NEWTON PITT, M.D.

C. H. GOLDING-BIRD, M.B.

E. C. PERRY, M.D.

W. H. A. JACOBSON, M.Ch.

L. E. SHAW, M.D.

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A. J. CLEVELAND, M.B.

Surgical Registrar and Tutor.

L. H. MCGAVIN, Esq.

Obstetric Registrar and Tutor.

G. BELLINGHAM SMITH, Esq.

Ophthalmic Registrar and Tutor.

A. W. ORMOND, Esq.

Warden of the College.

MR. DUNN.

Lying-in Charity.

DR. HORROCKS AND MR. TARGETT.

Dean of the Medical School.

DR. SHAW.

Sub-Dean.

DR. FAWCETT.

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<i>Clinical Medicine</i>	THE PHYSICIANS AND ASSISTANT PHYSICIANS.
<i>Clinical Surgery</i>	THE SURGEONS AND ASSISTANT SURGEONS.
<i>Medicine</i>	DR. TAYLOR AND DR. HALE WHITE.
<i>Practical Medicine</i>	DR. CLEVELAND.
<i>Surgery</i>	MR. HOWSE AND MR. LUCAS.
<i>Operative Surgery</i>	MR. FRIPP AND MR. STEWARD.
<i>Practical Surgery</i>	MR. MCGAVIN.
<i>Midwifery and Diseases of Women</i> ...	DR. GALABIN.
<i>Practical Obstetrics</i>	MR. TARGETT.
<i>Mental Diseases</i>	DR. SAVAGE.
<i>Ophthalmic Surgery</i>	MR. HIGGENS.
<i>Dental Surgery</i>	MR. NEWLAND-PEDLEY.
<i>Aural Surgery</i>	MR. LAIDLAW PURVES.
<i>Diseases of the Skin</i>	DR. PERRY.
<i>Diseases of the Throat</i>	MR. SYMONDS.
<i>Electro-Therapeutics</i>	DR. BRYANT.
<i>Anæsthetics</i>	MR. TOM BIRD AND MR. ROWELL.
<i>Hygiene and Public Health</i>	DR. SYKES AND MR. PAKES.
<i>Pathology</i>	DR. PITT.
<i>Morbid Anatomy</i>	DR. BRYANT AND DR. FAWCETT.
<i>Morbid Histology</i>	MR. BELLINGHAM SMITH.
<i>Surgical Pathology</i>	MR. FRIPP.
<i>Bacteriology</i>	DR. WASHBOURN AND MR. PAKES.
<i>Forensic Medicine</i>	DR. STEVENSON.
<i>Anatomy</i>	MR. LANE AND MR. DUNN.
<i>Practical Anatomy</i>	MR. FAGGE, MR. ROWLANDS AND MR. SWAN.
<i>Physiology</i>	DR. WASHBOURN AND DR. PEMBREY.
<i>Practical Physiology</i>	DR. PEMBREY, DR. BEDDARD AND DR. SPRIGGS.
<i>Materia Medica and Therapeutics</i> ...	DR. PERRY.
<i>Practical Pharmacy</i>	THE HOSPITAL DISPENSER.
<i>Chemistry</i>	MR. GROVES, F.R.S., AND MR. WADE.
<i>Practical Chemistry</i>	MR. WADE AND MR. RICHMOND.
<i>Experimental Physics</i>	PROFESSOR REINOLD, F.R.S., AND MR. RICHMOND.
<i>Biology</i>	DR. WILLEY, DR. STEVENS AND MR. SWAN.
<i>Psychology</i>	DR. SAVAGE AND DR. HYSLOP.

The Hospital contains 695 Beds, of which 544 are in constant occupation. Special Classes are held for Students preparing for the University, and other Higher Examinations.

APPOINTMENTS.

All Hospital Appointments are made strictly in accordance with the merits of the Candidates, and without extra payment. There are 28 Resident Appointments open to Students of the Hospital annually without payment of additional fees, and numerous Non-resident Appointments in the general and special departments. The Queen Victoria Ward recently re-opened will provide additional accommodation for gynaecological and maternity cases.

ENTRANCE SCHOLARSHIPS.

YEARLY IN SEPTEMBER.

Two Open Scholarships in Arts, one of the value of £100 open to Candidates under 20 years of age, and one of £50 open to Candidates under 25 years of age. Two Open Scholarships in Science, one of the value of £150, and another of £60, open to Candidates under 25 years of age. One Open Scholarship for University Students who have completed their study of Anatomy and Physiology, of the value of £50.

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Are awarded to Students in their various years, amounting in the aggregate to more than £650.

DENTAL SCHOOL.

A recognised Dental School is attached to the Hospital, which affords to Students all the instruction required for a License in Dental Surgery.

NEW SCHOOL BUILDINGS.

The new Theatre and Laboratories, opened in June, 1897, by H.R.H. The Prince of Wales, afford every facility for practical instruction in Physiology.

COLLEGE.

The Residential College accommodates about 50 Students in addition to the Resident Staff of the Hospital. It contains a large Dining Hall, Reading Room, Library, and Gymnasium for the use of the Students' Club.

For Prospectus and further information, apply to the Dean, Dr. SHAW, Guy's Hospital, London Bridge, S.E.

GUY'S HOSPITAL.

THE STAFF OF THE DENTAL SCHOOL. 1900.

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F. NEWLAND-PEDLEY, F.R.C.S., L.D.S.E.
W. A. MAGGS, L.R.C.P., M.R.C.S., L.D.S.E.
J. H. BADCOCK, L.R.C.P., M.R.C.S., L.D.S.E.

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H. L. PILLIN, L.D.S.E.	M. F. HOPSON, L.D.S.E.

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J. L. PAYNE, L.R.C.P., M.R.C.S., L.D.S.E.	E. B. DOWSETT, L.R.C.P., M.R.C.S., L.D.S.E.
C. J. HINCHLIFF, L.D.S.E.	P. S. CAMPKIN, L.D.S.E.

Anæsthetists.

F. W. COCK, M.D., M.S.	F. J. STEWARD, M.S., F.R.C.S.
H. F. LANCASTER, M.D.	W. S. HANDLEY, M.D., M.S.
C. J. OGLE, M.R.C.S.	R. P. ROWLANDS, L.R.C.P., M.R.C.S.

Lecturers.

Dental Surgery and Pathology.—Mr. NEWLAND-PEDLEY.

Dental Anatomy and Physiology.—Mr. MAGGS.

Operative Dental Surgery.—Mr. BADCOCK.

Dental Mechanics.—Mr. WYNNE ROUW.

Practical Dental Mechanics.—Mr. PILLIN.

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Metallurgy.—C. E. GROVES, F.R.S.

Practical Dental Metallurgy.—Mr. HOPSON.

Curator of Dental Museum.—Mr. PAYNE.

Dean.—Dr. SHAW.

Sub-Dean.—Dr. FAWCETT.

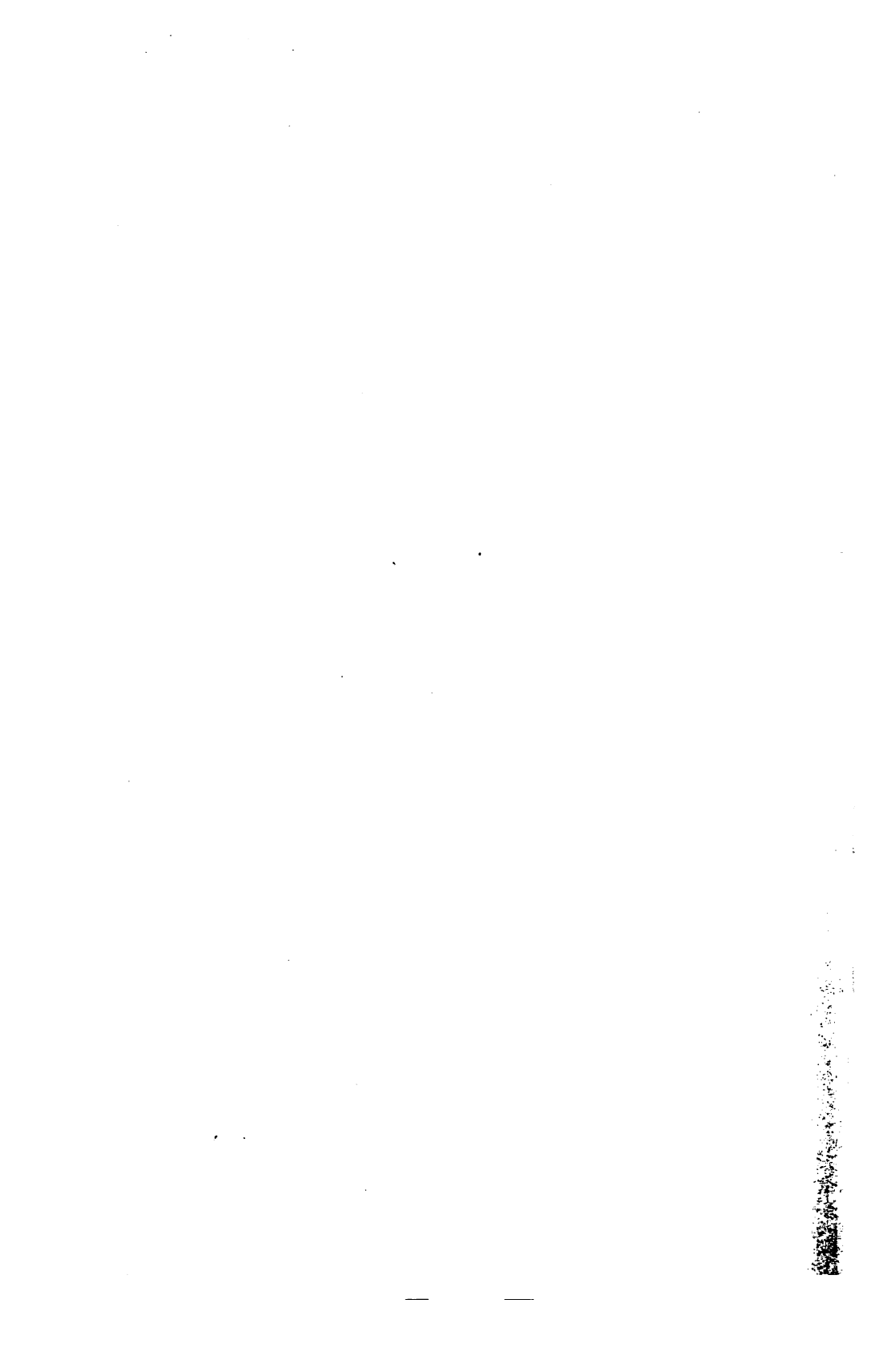
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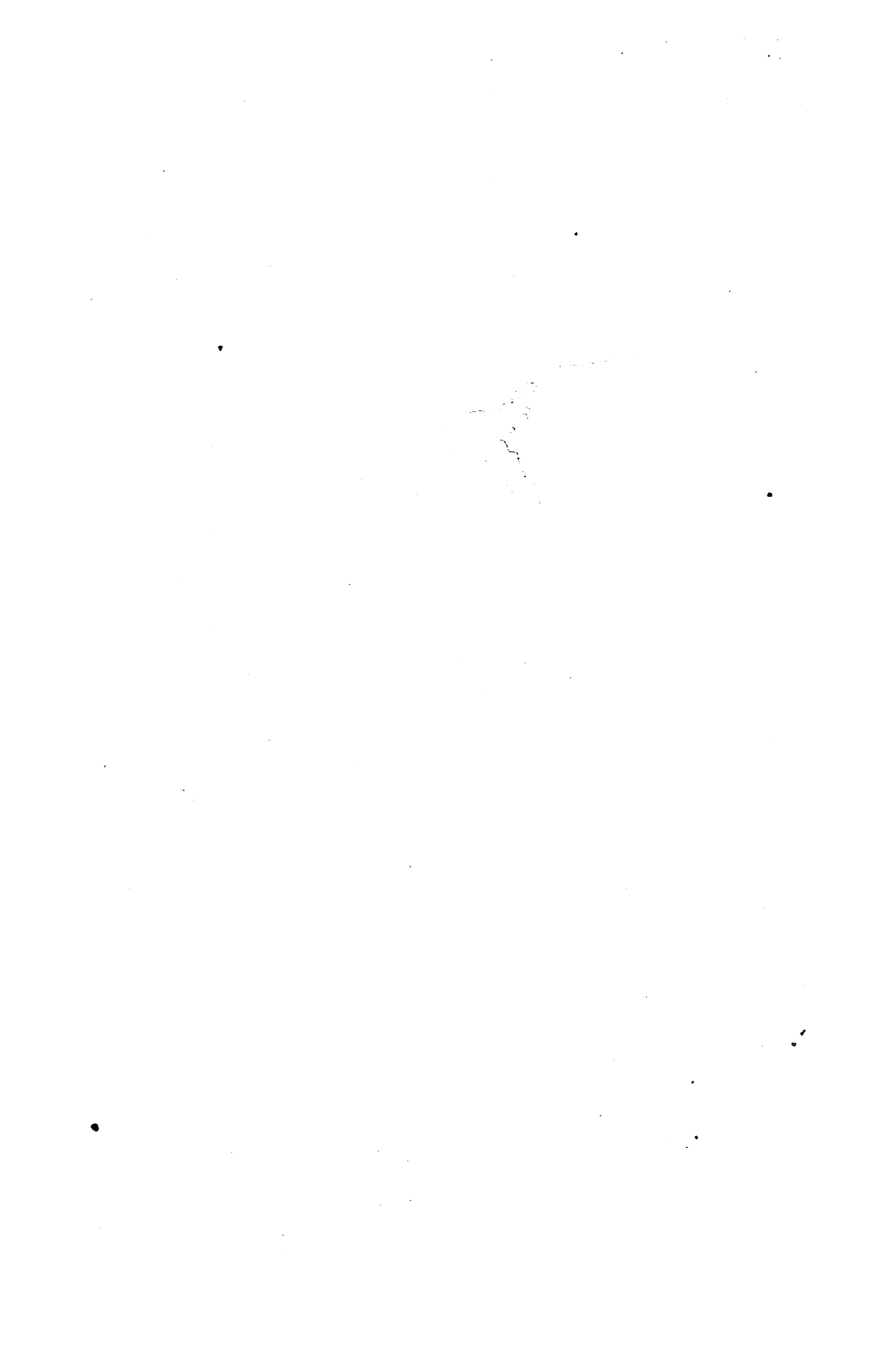
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 11. List of Preparations Recently Added to the Museum. By Lauriston E Shaw and E. Cooper Perry.
- List of Gentlemen Educated at Guy's Hospital who have passed the examinations of the several Universities, Colleges, etc., in the year 1896.
- Clinical Appointments held during the year, 1896.
- Dental Appointments held during the year, 1896.

J & A. CHURCHILL, Great Marlborough Street.







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